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EXTRAPLEURAL THORACOPLASTY IN THE PRESENCE OF CONTRALATERAL PNEUMOTHORAX*

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SINCE the inception of the procedure of extrapleural thoracoplasty, the presence of active tuberculous disease in the contralateral lung has been considered as a definite contraindication to this operation. Many times thoracoplasty has been delayed or the idea of employing this procedure completely abandoned because uncontrolled contralateral disease has been present. Throughout the years in which thoracoplasty has found an ever increasing range of usefulness, many persons have undoubtedly been denied its benefits because the so-called good lung was involved.

In the hope that more people could receive the benefits of adequate collapse therapy, contralateral pneumothorax simultaneously with that of extrapleural thoracoplasty was tried.

Bilateral collapse therapy is certainly not a new procedure. Ascoli,[1] as early as 1912, employed bilateral pneumothorax when even unilateral pneumothorax as a method of treatment was still new. Since that time various bilateral collapse combinations have been successfully used in the treatment of pulmonary tuberculosis. Strangely enough, thoracoplasty has not entered into these bilateral combinations until recently. Graham, Singer, and Ballon[2] state in their text that "to our knowledge the performance of thoracoplasty on one side with pneumothorax on the other has been done on seven occasions." A check of these cases reveals

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that in only two instances was pneumothorax present at the time the thoracoplasty was done. These were the cases of Samson[3] and Freund,[4] of Germany, and were reported in 1924 and 1931, respectively. Coryllos[5] reported the first large series of cases in 1936. His report was incident to a general discussion of thoracoplasty.

We are reporting the results of our experience with this method of treatment. We have employed it on eighty-five patients who had undergone 207 stages of thoracoplasty. These patients have been operated upon during the past five and one-half years. The type of thoracoplasty done in all cases has been that described by O'Brien[6] in 1933, namely, the resection of the entire first three ribs with their transverse processes at the first stage operation. The number of ribs removed at subsequent stages has been in accordance with the patient's ability to withstand further collapse therapy, and the need for further collapse has been estimated from the roentgenograms. Nitrous oxide-oxygen anesthesia has been used as the anesthetic in all cases reported.

Bilateral collapse therapy is certainly not a simple procedure. It is likewise not one to be entered upon lightly, especially when one-half of such collapse must be accomplished by a surgical procedure such as extrapleural thoracoplasty. Patients were picked on the basis of general well-being but were those in whom thoracoplasty was contraindicated by the presence of uncontrolled disease in the contralateral lung. It included, therefore, those cases with productive or mixed contralateral disease with cavity or with exudative disease with or without cavity. When the disease was exudative in type, the presence of cavity was not necessarily a prerequisite to the institution of pneumothorax for, as is well known, the course of the exudative type of tuberculosis is highly unpredictable. Aside from the state of the contralateral lung, other indications and contraindications were much the same as in the patient picked for thoracoplasty in whom the disease is unilateral.

The contralateral pneumothorax was instituted and maintained for varying lengths of time before thoracoplasty was started. In general, thoracoplasty was not started until it was certain that the pneumothorax would control the contralateral disease and that the respiratory reserve of the patient was such that thoracoplasty could be tolerated in its presence.

The degree of collapse on the contralateral side has varied with the need of the individual case. It has averaged approximately 20 per cent of the anatomic lung volume, although in certain individuals where the need for keeping a cavity closed has been great, the degree of collapse has been increased to as high as 35 to 40 per cent of the anatomic volume.

The postoperative care has been much the same as that employed for all thoracoplasty cases. There has been no routine use of the oxygen

tent in these cases, although where there was any doubt as to the patient's ability to withstand the added surgical procedure, such patients were immediately placed in the oxygen tent upon their return from the operating room. There can be no doubt that the timely use of oxygen does much to lighten the load on a cardio-respiratory combination, which at best must be sorely taxed.

RESULTS

Control of Disease in Contralateral Lung.—It will be seen by referring to Table I that thirty-seven patients of the total eighty-five, or 43.53 per cent, had exudative disease in the contralateral lung when pneumothorax was started. Twenty-three patients, or 27.06 per cent, had a mixed type of disease, and twenty-five patients, or 29.41 per cent, had productive disease. In other words, slightly over 70 per cent of the total group had a relatively acute form of tuberculosis present in the contralateral lung. Cavity was present in fifty-one of the eighty-five cases.

TABLE I

TYPES OF DISEASE IN THORACOPLASTY AND CONTRALATERAL LUNG

TYPE OF DISEASE	SIDE OF DISEASE			
	THORACOPLASTY SIDE		CONTRALATERAL SIDE	
	PATIENTS	PER CENT	PATIENTS	PER CENT
Exudative	27	31.76	37	43.53
Mixed	31	36.48	23	27.06
Productive	27	31.76	25	29.41
Total	85	100.0	85	100.0

Following establishment of pneumothorax on the side of the above mentioned lesions, the cavities were closed in all but five cases, which would place the cavity closure in this group at 90.2 per cent. The cavities varied in size from 1 to 5 cm. and were both solitary and multiple.

TABLE II

CAVITY STATUS BEFORE AND AFTER CONTRALATERAL PNEUMOTHORAX AND THORACOPLASTY

	CONTRALATERAL LUNG				THORACOPLASTY LUNG			
	BEFORE PNEUMOTHORAX		AFTER PNEUMOTHORAX		BEFORE OPERATION		AFTER OPERATION	
	PATIENTS	PER CENT	PATIENTS	PER CENT	PATIENTS	PER CENT	PATIENTS	PER CENT
Cavity open	51	100	5	9.8	84*		18	23.69
Cavity closed	0	0	46	90.2	0		58	76.31
Total	51	100	51	100.0	84		76	100.0

*Percentage of cavity closure figured on 76 cases. Cavity closure after thoracoplasty figured on a basis of 84 patients, 69.04 per cent.

The pneumothorax is still present in twenty-seven of the original eighty-five cases. In this group the contralateral disease is controlled in twenty-two patients but has increased under the pneumothorax and

since the thoracoplasty in five. In eight instances the pneumothorax was lost during the time when thoracoplasty was being done. Seven of these patients have shown no increase in their controlled disease; in one patient the disease has increased.

In the group (twenty-eight patients) in which the pneumothorax was purposely given up or lost at time intervals from one month to three years following thoracoplasty, there have been five cases in which the disease has extended. Among the ten patients who died late after thoracoplasty, six had extension of their disease in the contralateral lung.

TABLE III

PRESENT STATUS OF DISEASE IN CONTRALATERAL LUNG

PRESENT STATUS	TOTAL NUMBER	DISEASE CONTROLLED OR HEALED	DISEASE EXTENDED
Still present	27	22	5
Lost during operation	8	7	1
Lost or given up—1-3 mo.	6	4	2
Lost or given up—3-6 mo.	8	7	1
Lost or given up—6 mo. or more	17	15	2
Late death	10	4	6
Total	76	59 (77.63%)	17 (22.39%)

To summarize, it will be seen that in seventy-six cases, which include the nine immediate deaths, the disease in the contralateral lung was controlled in fifty-nine, or 77.63 per cent, and extended in seventeen, or 22.39 per cent. It is interesting, however, that no cavity developed in any of these areas of extension and that no cavity opened upon re-expansion of the lung.

TABLE IV

RESULTS IN 85 PATIENTS WHO HAD CONTRALATERAL PNEUMOTHORAX AT TIME OF THORACOPLASTY

STATUS	NUMBER PATIENTS	PERCENTAGE
Deaths (immediate)	8	9.4
Deaths (late)	10	11.7
Patients living	67	78.9
Total	85	100.0

TABLE V

COMPLICATIONS OF CONTRALATERAL PNEUMOTHORAX

COMPLICATIONS	PATIENTS	PER CENT
None	52	61.17
Serous fluid	22	25.88
Empyema, tuberculous	7*	8.24
Spontaneous pneumothorax	4†	4.71
Total	85	100.0

*Two patients died; one patient lost pneumothorax space as a result of empyema.
†One additional spontaneous pneumothorax in group with serous fluid.

Complications of the contralateral pneumothorax have been relatively few. Fluid was present in 25.88 per cent of the cases, but in only ten was it necessary to aspirate the fluid, and aspiration has been carried out in any case where the amount of fluid was large enough to be easily obtained. Seven patients developed a definite tuberculous empyema. Two of these died late after thoracoplasty of their empyema and generalized tuberculosis.

Spontaneous pneumothorax developed in four patients, but, while bothersome at the time, did not lead to serious consequence. It was necessary to insert a catheter into the pleural cavity of one patient in order to adequately cope with the emergency; the pneumothorax space was lost but the disease remained healed and the cavity closed.

Results From Thoracoplasty.—Cavity was present in eighty-four of the eighty-five patients undergoing thoracoplasty. The sputum was positive on direct smear in eighty-four patients, and positive only on culture in another where there was definite cavity demonstrated by x-ray film. Two hundred and seven stages were done and these varied in the individual case from one to five stages.

TABLE VI

SPUTUM STATUS AFTER CONTRALATERAL PNEUMOTHORAX AND THORACOPLASTY

SPUTUM STATUS	BEFORE CONTRALATERAL PNEUMOTHORAX	AFTER CONTRALATERAL PNEUMOTHORAX	AFTER THORACOPLASTY PATIENTS	AFTER THORACOPLASTY PERCENTAGE
Sputum positive	85	84	24	31.6
Sputum negative	0	1	52	68.4

There were eight immediate postoperative deaths or a mortality of 9.4 per cent. Cavities closed in fifty-eight of the seventy-six patients who survived the operation, or 76.31 per cent. Sputum was converted in fifty-two patients, or 68.4 per cent. Sputum has been recorded as positive in those cases where the weekly test and concentrates have not been negative for three consecutive months.

TABLE VII

PRESENT STATUS

STATUS OF TUBERCULOSIS	NO. PATIENTS	PERCENTAGE PATIENTS	STATUS OF PATIENT		ECONOMIC STATUS	
			LIVING AT HOME	LIVING IN HOSPITAL	ABLE TO WORK	UNABLE TO WORK
Arrested	19	22.30	18	1	19	0
Apparently arrested	8	9.4	4	4	2	6
Quiescent	13	15.2	0	13	0	13
Improved	23	27.1	2	21	0	23
Unimproved	2	2.3	0	2	0	2
Dead	18	21.1	—	—	—	—
Untraced	2	2.3	—	—	—	—
Total	85	99.7	24	41	21	44

The term late results may not be wholly justified because some of the cases appearing in the group have been done relatively recently.

At present, of the entire group, nineteen cases are arrested and the patients are working or able to work. Eight are apparently arrested, and of this group two are working. Thirteen are quiescent. Twenty-three are improved. Two are worse. Eighteen are dead, giving a total mortality of 21.1 per cent. Two are untraced.

DISCUSSION

It would seem from the results recorded in both charts and the text that the use of pneumothorax as a therapeutic measure in the treatment of the disease in the contralateral lung simultaneously with the performance of thoracoplasty is a justifiable and useful procedure. Certainly the patient with a thoracoplasty lesion in one side but in whom the state of the contralateral lung contraindicates such a procedure has little to look forward to. While untreated lesions of this type do heal, such experience is not the rule. Werner and O'Brien[7] showed many years ago that the tendency is for cavities to increase in size rather than decrease. The mortality studies of Barnes and Barnes[8] give added weight to this contention. Such experience, therefore, minimizes the chance of adequate healing in the contralateral lung, especially if the spread has been large and cavity is present. Not infrequently, waiting for disease to heal in the contralateral lung without treatment has resulted in increase of the disease in both sides.

The work of Brosius and Birkelo,[9] and of Wood and Miller,[10] which clearly shows that even small amounts of tuberculous disease do irreparable damage to the pulmonary circulation and thus decrease the efficiency of the lung as an oxygenating unit, gives added reason for control of the contralateral disease. It seems very likely that extensive disease with its subsequent scarring does much more to injure the oxygenating function of the lung than does a considerable amount of pneumothorax (Whitehead and Miller;[11] Whitehead, Tuttle, and Miller[12]). It is thus highly essential to control the contralateral disease even if thoracoplasty is not immediately contemplated.

This group of patients reported were far from being what is ordinarily considered as good risks. As a whole they were exceedingly poor risks. In the first place, they all had far-advanced pulmonary disease, which of itself depletes the general constitutional reserves. In the second place, there was added burden of a contralateral pneumothorax. Over two-thirds of the patients had exudative or mixed type disease on both sides. This, as is well known, definitely increased the hazard of thoracoplasty. Furthermore, there were eight cases, or approximately 10 per cent of the group, in which the vital capacity was under 1000 c.c.

The completion of the thoracoplasty was impossible in ten patients, and of this number, six are now dead. This number may be compared

with twenty-five cases in which completion was impossible out of a series of 455 patients undergoing thoracoplasty, recently studied by O'Brien, Day, Chapman, and Tuttle.[13]

Despite these quoted facts, the results in general have been, we feel, most encouraging. There was, as has been previously noted, an early mortality of 9.4 per cent and a late mortality (which will increase as a result of the recency of some cases) of 11.7 per cent. These figures compare most favorably with those of O'Brien, Day, Chapman, and Tuttle, who found a mortality of 9.5 per cent and 20.7 per cent, respectively, for the early and late groups. To the authors, the fact that from such a group 31.6 per cent are arrested or apparently arrested, alone justifies the means.

It should be borne in mind that the handling of such cases requires extreme care. We have found that our results have been better when air is given relatively often (4 to 5 days) and in small amounts (100 to 250 c.c.). This tends to maintain the degree of pneumothorax at a constant level and does not subject the patient to marked fluctuation in pulmonary volume. It also keeps the lung at a constant distance from the chest wall and minimizes the danger of loss of the pneumothorax and of spontaneous pneumothorax.

It has been the practice of some clinics to proceed with thoracoplasty when there is a moderate amount of disease in the contralateral lung. Following the thoracoplasty there is an attempt made to control the contralateral disease. Such practice, we believe, is not a good one, but because it does not seem right to subject a patient to an operative procedure such as thoracoplasty when it is not definitely known that contralateral pneumothorax can be established and the lesion controlled. Furthermore, the dangers incident to establishing a pneumothorax are greater following thoracoplasty, especially if the latter has been an extensive procedure. Spontaneous pneumothorax is a much more serious accident when the opposite lung is collapsed in part or completely by thoracoplasty. Also, if adhesions which require cutting are present, this can be done with greater safety if the opposite lung is present as a functioning unit, even if such function is far from complete.

When is the optimum time for doing thoracoplasty in such cases? This, we believe, depends to a large extent on the amount of disease present. If the amount is small and the contralateral pneumothorax is being done as a precautionary measure, then it is necessary to wait only until the pneumothorax has been stabilized. Such stabilization is important, for in a pneumothorax such as these where the mantle of air is small, the danger of losing the air space is great if the absorption of air is abnormally rapid, as is often the case in a new pneumothorax. Of the eight patients in this series who lost their space during thoraco-

plasty, six of them were of this type. We have, therefore, come to believe that the pneumothorax should be relatively stable before thoracoplasty is started.

If the amount of disease is large or moderately large, it is necessary to wait until some clearing has taken place; however, clearing does not need to be complete, nor does healing of the lesions have to be completely accomplished. In some cases it may be necessary to carry for some time a rather large pneumothorax (40 to 50 per cent), and then when it appears that the lesion has cleared sufficiently, the amount of pneumothorax is decreased and thoracoplasty started. It is better to do the thoracoplasty, of course, with as small a contralateral pneumothorax present as seems consistent with the ability to hold the space and control the disease process.

In some of the early cases we proceeded too rapidly to thoracoplasty, with the result that the space was lost, as has been mentioned, or the thoracoplasty had to be left incomplete because of inadequate pulmonary reserve. This was true especially in the exudative type of disease. In such patients, the dangers of fluid and of spontaneous pneumothorax are always greater than in the more proliferative types of disease. It is best, therefore, to know that enough proliferative change has taken place in the contralateral lesion to forestall such dangers. Furthermore, moderate clearing of exudative disease increases the functioning lung volume and this is of great importance in patients whose lung volume is considerably impaired.

Where cavities are present it is not necessary that they be completely closed, but there should be good assurance that closure can be accomplished when the thoracoplasty has been completed and it is again possible to carry a larger pneumothorax.

To summarize this important point, the optimum time for thoracoplasty is when it becomes apparent that the contralateral disease can be controlled or when the cavities are closed or closing. If no cavity is present, the contralateral disease should show evidence of clearing. If the amount of disease is large, it is better to have considerable clearing before thoracoplasty is started. Exudative disease without cavity often clears rapidly under a mantle of air, but from the standpoint of respiratory reserve, the dangers of fluid, and of spontaneous pneumothorax, it is best that such clearing has taken place before thoracoplasty is attempted.

On the other hand, thoracoplasty must not be delayed too long. It would naturally be assumed that a pneumothorax of long standing might lend itself better to such treatment than a relatively recent pneumothorax. This, from the standpoint of healed disease, may be true, but it must be borne in mind that an old pneumothorax is also labile, and, if the need to rapidly re-expand the lung arises, as it occasionally does, duration of pneumothorax is an important factor.

Three of the eight deaths in this series were patients in whom pneumothorax was of long standing, and when the need for increased lung volume arose, rapid re-expansion was found to be impossible.

A large number of the patients who comprise this group are still in the hospital. A number of these might, under ordinary circumstances, be home. However, we routinely keep our postoperative thoracoplasties at strict bed rest for from eight months to one year following their last stage. Surgery must never be considered as a substitute for bed rest. From autopsy specimens, we are of the belief that it requires at least eight months for a cavity to heal under the permanent collapse of thoracoplasty. The relatively small late mortality, we feel, may be due in part to adequate postoperative hospitalization.

CONCLUSIONS

1. Eighty-five patients who were subjected to 207 stages of thoracoplasty have been reported.

2. There was an immediate mortality of 9.4 per cent and a late mortality of 11.7 per cent.

3. The contralateral pneumothorax was complicated by simple fluid in 25.88 per cent of the cases, by empyema in 8.24 per cent of cases, and by spontaneous pneumothorax in 4.71 per cent of cases.

4. Twenty-seven cases are arrested or apparently arrested; thirteen are quiescent; twenty-three are improved; two are unimproved; eighteen patients are dead.

REFERENCES

1. Ascoli, M.: Quoted from Surgical Diseases of the Chest, Philadelphia, 1935, Lea & Febiger.
2. Graham, E. A., Singer, J. J., Ballon, H.: Surgical Diseases of the Chest, Philadelphia, 1935, Lea & Febiger.
3. Samson, J. W.: Quoted from Surgical Diseases of the Chest. By Graham, Singer, and Ballon, p. 1039, Philadelphia, 1935, Lea & Febiger.
4. Freund, A.: Quoted from Surgical Diseases of the Chest. By Graham, Singer, and Ballon, Philadelphia, 1935, Lea & Febiger.
5. Coryllos, P. N.: Personal Communication.
6. O'Brien, E. J.: J. THORACIC SURG. 3: 159, 1933.
7. Werner, W., and O'Brien, E. J.: Am. J. Roentgenol. 24: 626, 1930.
8. Barnes, H. C., Barnes, L. R. P.: Am. Rev. Tuberc. 18: 412, 1928.
9. Brosius, W., and Birkelo, C. C.: In press, Am. J. Radiology.
10. Wood, D. A., and Miller, M.: J. THORACIC SURG. 7: 649, 1938.
11. Whitehead, W. K., and Miller, A.: Proc. Soc. Exper. Biol. & Med. 38: 991, 1938.
12. Whitehead, W. K., Tuttle, W. M., and Miller, A.: To be published.
13. O'Brien, E. J., Day, J. C., Chapman, P., and Tuttle, W. M.: To be published.

GIANT TUBERCULOUS CAVITIES OF THE LUNG

Pathogenesis, Pathologic Physiology, and Surgical Treatment*

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IN THE course of pulmonary tuberculosis a number of tuberculous cavities present a considerable increase in size. Their diameter reaches 8 to 10 cm. or more. They are designated as giant cavities.

These cavities may develop very rapidly, sometimes within a few weeks or even a few days (Izzo, Aguillar, and Shiffrin,[69] Bernou and Vivant[19]). They present peculiar variations of size; they may decrease and again increase or even disappear completely and spontaneously, without any apparent reason (Morin and Waitz,[97] Korol,[81] Jaquerod,[76] Hudson,[66, 67] Minet and Patoir,[96] Pagel and Robinson,[107] Katz,[79] Mantoux,[93] Sisti,[134] and Bernard[17]). Often they are perfectly spherical cavities with thin walls; in other cases their walls are thick; but even the thickness of their walls may undergo marked changes. A number of these cavities are empty; others contain fluid limited by a fluid level. Again, this fluid may completely fill up the cavity which is then changed to a circular opaque shadow occupying in the radiogram the place where the cavity was previously seen. This fluid often appears and disappears successively in the same cavity. This phenomenon has more especially attracted the attention of a number of authors who have coined for these cavities the name of "blocked cavities" (Salkin, Cadden and McIndoe,[122] Eloesser[46]).

Giant cavities may be accompanied by no physical signs (silent cavities) or may present amphoric resonance and be confused with pneumothorax. They may be perfectly latent and only accidentally discovered in patients apparently in perfect health (Huriau[68]). They may remain stationary for a number of years with periods of good health interrupted by periods of cough, expectoration, and septic symptoms. They may be located in part of a lobe, more frequently, the apices of the upper or lower lobes; but they may also occupy one entire lobe which seems completely sloughed out and replaced by the cavity. Less frequently they may occupy more than one lobe (multilobar giant cavities of Ameuille, Coste, and Le Melletier,[10] Courcoux and Lermoyer,[40] Roubier,[121] D'Hour and Delcour[45]). These multilobar cavities may appear spherical or flattened ("Cavernes Céantes en Lames" of Ameuille and Le Melletier[11]). Such multilobar cavities often communicate through large holes bored in the interlobar fissures. Their

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evolution also presents the most capricious course. A number of them remain stationary for years (Huriau[68]), while others present a steadily progressive course terminating in rapid death.

The knowledge of giant cavities is by no means a recent acquisition. Ameuille[6] is studying the history of tuberculous cavities since Laënnec (1933) reminded one that Laënnec[84] had mentioned giant cavities which were diagnosed during life and checked by autopsy. Peter (1897) reported a case of a young woman with signs of giant cavity which healed spontaneously. Jaccound[71] (1881) has insisted on the curability of giant cavities. Katz[79] has given an excellent history of these cavities.

However, the question of giant cavities has grown in importance since the advent of radiography, which has rendered possible the accurate following of their changes, and more especially since the generalization of collapse treatment. In fact, whereas these cavities may disappear spontaneously, they often oppose considerable resistance to pneumothorax and even to thoracoplasty or they even grow larger in the artificially collapsed lung.

During the last decade and more especially during the last five years a considerable number of publications have appeared in the continent and in this country on this question. The discussion has shifted from pure morphologic and pathologic grounds to the pathologic physiology and mechanics of these cavities. Ameuille[9] in his paper on the draining bronchi of tuberculous cavities (1927) has attracted the attention of phthisiologists to the importance of the latter upon the evolution of the cavities and formulated the hypothesis that "enlargement of cavities was due to closure of these bronchi and subsequent retention of secretions in them." Coryllos[29, 32] (1933, 1936) has elaborated a completely contrary conception and supported the thesis that "narrowing of the draining bronchi caused ballooning of cavities by the mechanism of obstructive emphysema, whereas complete and definite obstruction of the draining bronchi caused the disappearance of cavities by the mechanism of obstructive atelectasis." Ornstein,[106] on the same ground, designated the former cavities as ball or check valve cavities.

At present, authors are divided into two camps. The first group follow the opinion of Ameuille and consider that closure of a draining bronchus hampers the cure of cavities. The second concur in the opinion of Coryllos and believe that complete closure of these bronchi is the only mechanism by which tuberculous cavities may heal, whatever their size and the nature of their walls may be, even when anatomically they cannot collapse.

In the present paper we shall present the results of our more recent studies on giant cavities. These studies are based (1) on direct examination of these cavities by means of a specially constructed thoracoscope, designated as a cavernoscope, which is introduced into the cavity through the chest wall; (2) on serial gas analysis of the air contained

in these cavities and on the study of the pressure therein; (3) on their clinical study; (4) on surgical attempts at closure of these cavities and especially the ones that have resisted thoracoplastic collapse; (5) on bacteriologic studies of the content of these cavities, taken from cavities opened aseptically during surgical operations.

1. DIVISION OF CAVITIES: TENSION CAVITIES

It is our contention that giant cavities should be recognized as belonging to two varieties. First, cavities presenting positive pressures of the air contained in them, and second, cavities presenting atmospheric pressures. There is a third variety in which pressures are alternatively positive or atmospheric. It is not necessary to reserve a special place for the latter because their characters vary in accordance with their pressures, so that these cavities behave as do the first or the second variety of giant cavities, according to the status of their intracavitary pressures.

The difference between these two forms of cavities is fundamental. (1) *Cavities with positive pressures (tension cavities)* are always spherical, and they correspond to the variety known as ballooning, check or ball valve cavities. Their walls are thin, the sputum is often negative, the general condition of the patient, rather good. As a rule they show marked changes in their size; fluid is often present in them. These cavities may disappear spontaneously, either permanently or temporarily, or even disappear and reappear periodically at the same place and with the same characteristics. Lipiodol and dyes injected into these cavities through the chest wall remain in them for a variable length of time but are finally expectorated. (2) *Cavities with pressures equal to the atmospheric pressure,* on the contrary, show no accumulation of fluid, do not disappear spontaneously, and present unyielding resistance to collapse treatment. Moreover, their walls are more frequently thick and the sputum is persistently positive. Lipiodol and dyes injected into them are immediately or very rapidly expectorated. These cavities increase in size gradually and steadily by continuous sloughing of the pulmonary parenchyma. They correspond to the type of chronic progressive cavities of Ornstein and Ulmar.[106]

The cavities of the first variety which we shall call "tension cavities," because positive pressure in them is their more important character, may change into the second variety. As their positive pressure becomes atmospheric, fluid previously retained in them disappears and is expectorated; sputum becomes positive and the general condition of the patient declines. Concurrently, marked changes take place in their walls which become thicker. Conversely, if the intracavitary pressures again become positive, a regression of the phenomena enumerated above takes place. Fluid accumulates again, the general condition of the patient improves, sputum becomes less positive or even negative, and the walls lose their thick appearance.

As an example we shall give here the case reported by Morin and Waitz[97] which presented six such cycles within seven months. This case is typical and fully representative of a considerable number of cases already published. We have and certainly every phthisiologist has observed similar cases.

A white girl, 23 years old, was admitted in the hospital (Leysin, Switzerland), Dec. 27, 1925. Onset of the disease occurred January, 1925, with hemoptysis. In 1921 she had a grippe for which she remained in bed for two weeks. Since then she was coughing. On admission, sputum was positive (Gaffky 7) and moist râles were present on the right chest at the angle of the scapula. X-ray examination showed a thick wall cavity at the apex of the right lower lobe and an acinous infiltration in the left lower lobe. Gradually the general condition of the patient had improved, temperature became normal, weight increased, and sputum became negative on concentration; clinical signs disappeared. In x-ray films taken in September, 1926, the cavity was no more visible, its walls were hardly outlined, and the lesions in the left side had improved; sputum again became negative. In February, 1927, again temperature rose to 102° F., the weight decreased, and sputum became positive. X-ray examination showed that the initial cavity had appeared again; it was larger than before and it had markedly thick walls. Râles were present. Within twenty-five days the patient again improved considerably. Sputum again became negative, and x-ray films of Aug. 8, 1927, showed the cavity smaller, and with thin walls. These cycles of large thick-walled cavity with positive sputum and septic condition followed by clinical improvement, negative sputum, disappearance or marked decrease of the size of cavity and considerable changes in the thickness of its walls were repeated six times in seven months (April, 1927, July, 1927, August, 1927, and December, 1927). In June, 1928, the patient left the hospital with negative sputum.

Similar cases have been reported by Bernard, Herrensmidt and Morin,[17] Korol,[81] Mantoux[93] (Mantoux called these cavities "accordion cavities"), Salkin, Cadden, and McIndoe,[122] Bernou and Vivant,[19] Katz,[79] Eloesser,[46] and others.

As it will be elaborated later, during the periods of recrudescence of clinical symptoms (elevation of temperature, reappearance of cavities, thickening of their walls, and positive sputum), the intracavitary pressure drops and becomes near atmospheric. At the same time the clinical composition of their gaseous content becomes almost the same as of the atmospheric air. It is obvious, we believe, that the one possible explanation of these variations is the variation of the degree of patency of the draining bronchus of these cavities. Only wide patency of the draining bronchus can explain atmospheric pressure in the cavity, absence of retention, and positive sputum; conversely only partial obstruction of this bronchus with valve action, allowing ingress of air during inspiration but not free egress during expiration, can explain positive pressure in the cavity, retention of exudate or of fluid injected into it through the chest wall and negative sputum. However, it is obvious that the fact that these cavities remain open indicates that this obstruction is only partial and transient. Otherwise if obstruction was complete positive pressure in the cavity would not be possible. Therefore, as will be shown later, the term "blocked cavities" used by the authors

mentioned above[122, 46] is improper and misleading; it indicates only a temporary status of the draining bronchus of the cavity but fails to indicate whether this bronchus is really closed or open, the only factor which regulates the course of these cavities. Moreover, this term does not designate any distinctive variety of cavities but only a temporary phase in the evolution of any tuberculous cavity. It is, therefore, a useless and confusing term adding nothing to our knowledge of these cavities. On the other hand, we actually believe that the characters of the walls upon which classification of cavities has been based (Pinner,[109] Pottenger,[113] Jaquerod,[75] etc.) also vary according to the degree of patency of draining bronchi; patent bronchus allows free circulation of air into the cavities and, consequently, of adequate amount of oxygen which is necessary for the growth and the metabolism of the strictly aerobic tubercle bacilli. It is obvious that under these favorable conditions of growth and metabolic activity of tubercle bacilli, tuberculin is produced freely, causing increased tissue sensitivity and consequently perifocal reaction and infiltration and thickening of the walls of the cavities.

Thus, in the last analysis, all giant cavities are open cavities. In a number of them a valve mechanism is formed in their narrowed bronchus causing its intermittent and partial closure leading to the formation of positive pressure in these cavities (tension giant cavities). In others, on the contrary, the draining bronchus remains open (open giant cavities). Likewise all tuberculous cavities ought to be distinguished as open (continuously or intermittently) and closed cavities. The latter are cavities which shrink and disappear if the nature of their walls allows it, but even when they cannot shrink they may heal biologically (cavities with smooth walls) even when they remain visible in the x-ray films. On the contrary open cavities remain active unless their bronchi become completely and definitely obliterated, when they are changed to closed cavities. There is no room, we believe, for establishing any other variety of cavities. The term "blocked cavities" introduces an unnecessary subdivision in the already confused classification of tuberculous cavities; we shall take this point up again later.

The data and findings upon which this conception is based are reported as follows.

2. CAVERNOSCOPIC FINDINGS

So far the inner aspect of these cavities and their draining bronchi had been studied, exclusively, on post-mortem specimens. The identification of the draining bronchi in them is occasionally easy. Most often, however, it becomes very difficult or even impossible. More especially the degree of their patency cannot be ascertained with the accuracy commensurate with the importance of the problems involved. Therefore, we thought it necessary to devise a technique which could render possible the study of the internal aspect of giant cavities on the living

patient without harm to the latter. The details of the technique devised and used for that purpose will be elaborated upon in a forthcoming paper. In brief this procedure consists in the introduction through the chest wall of a thoracoscope, of very small diameter, especially constructed for that purpose and designated as a cavernoscope. This can be done without any more danger to the patient than a simple needling of the cavity.

After the cavity is accurately localized in anterior-posterior and posterior-anterior radiograms, and its contours marked on the skin of the patient under the fluoroscope, a needle, connected with a three-way stopcock and a 10 c.c. syringe is slowly introduced into the cavity through the chest wall nearest to the cavity. The introduction of the needle is done under constant aspiration in order to ascertain that neither free pleural cavity nor healthy lung is present between the chest wall and the cavity. The entrance of the needle into the cavity is announced by free aspiration of air into the syringe and often the aspiration of pus. Thus the exact depth of the cavity can be accurately determined. A reading of the intracavitary pressure is taken at this time and a sample of air is aspirated for gas analysis. The stopcock is closed and the needle is left in place; the small trocar and cannula of the cavernoscope are then introduced parallel to the needle. The trocar is withdrawn and the cannula left in place; in order to make certain that the cannula is in the cavity, another sample of air is taken for analysis and a new reading of pressures is made. These should be identical with the ones previously obtained through the needle; otherwise the experiment is not considered as sufficiently accurate.

The cavernoscope with its light carrier is now introduced through the cannula into the cavity. It is surprising with what facility the inside of the cavity can be explored by this simple procedure and the wealth of information which can be gained. From our experience we can state that in all cases in which intracavitary pressure was equal or almost equal to atmospheric, the orifice of the draining bronchus could be visualized. This was not always easy because of the irregularities and anfractuosities of the inner wall of the cavum and of the caseous material covering it. When the outlet could not be seen, slight suction applied through the needle revealed its location by the bubbling of air through it. In large cavities with thick walls we found that as a rule only one bronchial outlet was present or it was much larger than the others and represented the principal draining bronchus of the cavity. In all cavities of the upper lobe (the only ones explored so far) the bronchial outlet occupied the lower part of the cavity. Saline solution introduced into the cavity remained in it, at least for a while, even when the orifice of the draining bronchus was well visible and apparently patent. Injection of air through the needle into the cavity, causing increase of the intracavitary pressure, was unable to force the saline out

of the cavity. On the contrary, when suction was applied, air would bubble through the saline solution covering the bronchial outlet, and almost immediately the solution would flow through it into the bronchial tree and disappear from the cavity. It appears, therefore, that positive pressure closes the orifice of the bronchial outlet whereas suction opens it. This experience corroborates the findings of Eloesser,[46] and offers a rational explanation of them. This author in his study of the so-called blocked cavities had found that introduction of a drain into the cavity and suction of its air caused dye and lipiodol, previously introduced into the cavity and retained in it for several days, to pass into the bronchial tree. It is no wonder, therefore, that this author who attempted this suction with the idea in mind of producing collapse of these blocked cavities by aspiraitng the air entrapped in them not only failed to collapse the cavities but, on the contrary, caused the blocked bronchus to open, contrary to his expectations.

In all cases in which a large and patent bronchial outlet was present, the cavity expanded during inspiration and retracted during expiration, provided that the glottis was maintained open. On the contrary, very little contraction was noted during cough, or forcible expiration with glottis closed. This phenomenon is in perfect accordance with the mechanics of these cavities, as will be shown in the discussion of their pathologic physiology.

In three large cavities a second trocar cannula was introduced through the chest wall parallel to the cannula of the thoracoscope. This second cannula served for the introduction of a fine ureteral catheter by means of which the content of the cavity could be aspirated for bacteriologic examination, and also for ascertaining the patency of these outlets by introducing the tip of the catheter into them and injecting a few drops of gentian violet. The touching of these outlets caused cough, and the dye immediately appeared in the expectorated sputum.

This investigation was carried out in eight cases. In six large cavities with thick walls pressures were equal or almost equal to atmospheric and patent outlets were found. In two cases of thin-walled giant cavities with positive pressures we could not distinguish any orifices. But even in these, bubbling occurred when suction was applied through the needle. Thus, none of our eight cases was a case of really closed cavity, although in one (Fig. 1) lipiodol had remained in the cavity for over seventy-two hours. These findings show that the so-called blocked cavities are only apparently closed but actually open cavities. The confusion of the terms blocked and closed cavities explains, we believe, the misgivings of the investigations of Salkin et al. and of Eloesser.

We shall report here briefly two of our eight cases.

CASE 1.—J. B., a white male, 43 years old, was admitted to the Castle Point Veterans' Hospital, November, 1936. Onset of the disease occurred fifteen months prior to admission. Pneumothorax had been unsuccessful. On x-ray examination

the patient presented a giant cavity in the left apex, measuring 10 by 9 cm., with rather thin walls. A draining bronchus could be seen in the film (Fig. 1). No fluid level was present. The patient expectorated 4 to 6 ounces in twenty-four hours, especially during the morning. Sputum was strongly positive for tubercle bacilli (Gaffky 4 to 9).

Nov. 11, 1937, the cavity was carefully localized by radiograms and fluoroscopy. Under local anesthesia the cavity was needled with 19 gauge and 3 inch needle.

Fig. 1. Fig. 2.

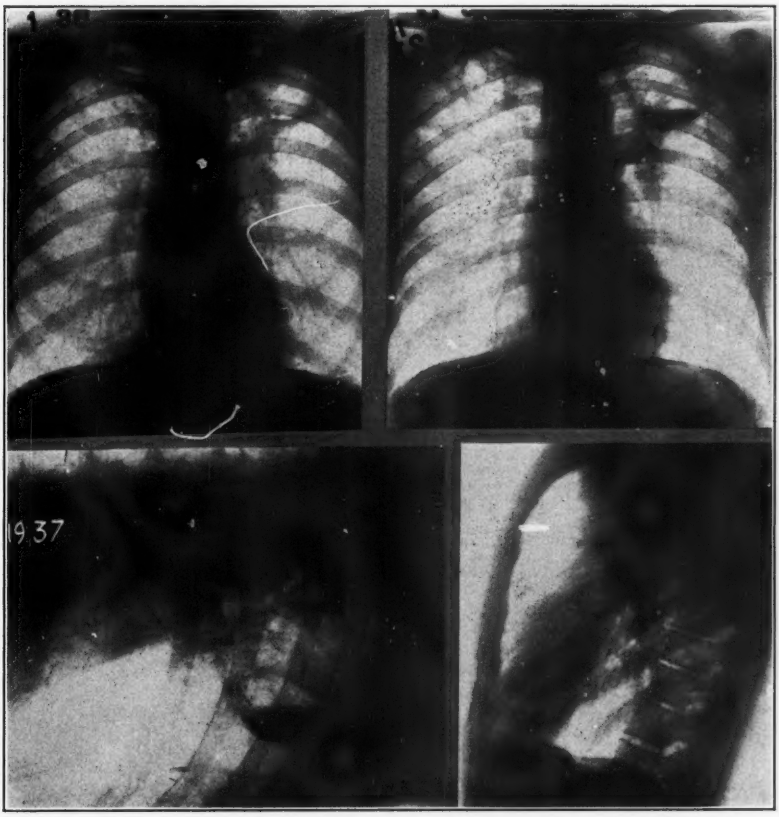

Fig. 3. Fig. 4.

Fig. 1.—Case 1. (J. B.) Giant cavity in the left lung. The cavity measures 10 by 9 cm. Note that there is a very small fluid level and the draining bronchus is well outlined.

Fig. 2.—Case 1. Retention of lipiodol and methylene blue solution in the giant cavity described in Fig. 1. The injection was made directly into the cavity through the thoracic wall. Note the two fluid levels, the lower darker one due to the lipiodol, and the upper lighter one due to the methylene blue. The draining bronchus is not outlined.

Fig. 3.—Case 1. X-ray picture taken in the left lateral position, the patient lying on his left side. Note the two fluid levels caused by the lipiodol and the methylene blue solution.

Fig. 4.—Case 1. X-ray picture taken in a left lateral erect position demonstrates the posterior location of the giant cavity.

Readings were -5 +6. Gas analysis showed O_2 19, 2. CO_2 0.4 per cent, that is, almost the same composition as of atmospheric air, with slightly less oxygen and higher carbon dioxide. This shows that during the incomplete closure of the draining bronchus, gas exchanges take place in the cavity.

The same study was repeated Nov. 19, 1937. The intracavitary pressures and gas analysis were the same as previously. Fifteen c.c. of lipiodol and 10 c.c. of methylene blue solution were injected into the cavity. Fig. 2 shows very clearly

Fig. 5. Fig. 6.

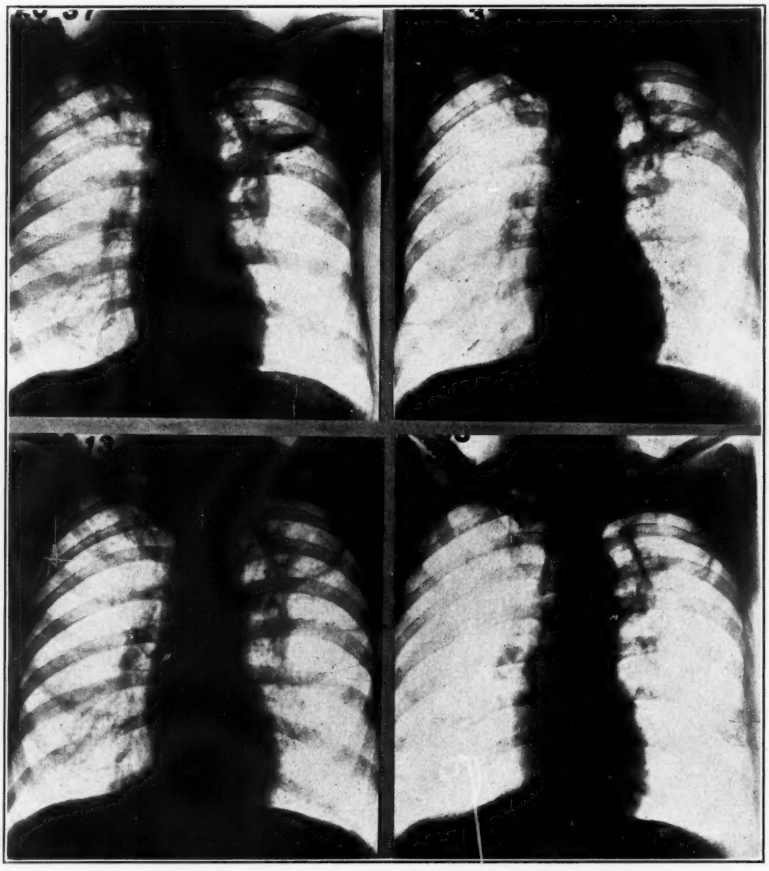

Fig. 7. Fig. 8.

Fig. 5.—Case 1. X-ray taken a day later than the films in Figs. 2, 3, and 4, demonstrating that the fluids injected into the giant cavity have not been evacuated.

Fig. 6.—Case 1. X-ray taken a day later than the x-ray, Fig. 5; it demonstrates the almost complete emptying of the lipiodol, after being present for seventy-two hours.

Fig. 7.—Case 1. A day later than the x-ray film, Fig. 6. Note that almost all the lipiodol is gone, and the fluid level present the day before has vanished.

Fig. 8.—Case 1. Taken fifty-three days later than the x-ray film, Fig. 7. Note that the giant cavity has no fluid level and the draining bronchus can easily be identified. This is an excellent example of an intermittently open and closed cavity.

two fluid levels, one dark due to lipiodol and the other lighter due to the methylene blue. Fig. 3 shows the same levels on a film taken in lateral left decubitus and Fig. 4 in erect lateral left exposure. Lipiodol and dye remained in the cavity for about forty-eight hours. They were still present November 20 (Fig. 5), but they were both eliminated November 21 (Fig. 6) and 22 (Fig. 7), and lipiodol could be seen in the bronchial tree. Three weeks later (Jan. 14, 1938) the aspect of the cavity was the same as before introduction of the lipiodol (Fig. 8). This cavity presented all the characters of "blocked cavity" of Salkin and of Eloesser.

Cavernoscopy was performed in this cavity a few days later. The internal surface of the cavity was irregular and covered with thick pus, swarming with tubercle bacilli. The orifice of the main draining bronchus was easily visualized. Saline injected into the cavity remained in it even when injection of air through the needle raised the pressure in the cavity to +20 cm. of water. On the contrary, when suction was applied, decreasing the intracavitary pressure to –10 cm. of water, air bubbled through the bronchial orifice and the saline suddenly disappeared. Through a second cannula introduced into the cavity, the orifice of the draining bronchus was cauterized with a few drops of 60 per cent silver nitrate solution. This patient did not show any untoward effect following this procedure. Nor did the application of silver nitrate on the bronchus cause any narrowing or closing of it.

CASE 2.—R. P., a 33-year-old white woman, was admitted at the Metropolitan Hospital, New York, Sept. 9, 1937. Onset of the disease was in 1935. Sputum was persistently positive. On admission (Fig. 9) the patient presented bilateral lesions with a spherical giant cavity in the right apex measuring 7.5 cm. in diameter, with rather thick walls and slight fluid level. On the left side there were two cavities, one rather small (4 cm.) and one very large (13 cm.), the latter with rather thin and irregular walls (Fig. 9). Two months later both cavities appeared larger, especially the one of the left apex, but the walls of the right cavity appeared less sharply defined and thinner (Fig. 10); three months later (Oct. 11, 1937) the right cavity showed a marked fluid level and had decreased in size, while the left was markedly increased and its walls were thicker (Fig. 11). Two months later (Dec. 10, 1937) the right cavity had completely disappeared while the left had further increased (Fig. 12); fluid level was present in the latter.

This case is of particular interest because it shows in the same patient the evolution of a cavity with closed bronchus (right lung) and of a cavity with intermittently open bronchus (left lung).

Cavernoscopy was performed on the left cavity on Feb. 14, 1938. Intracavitary pressure was –5 +6 with large oscillations. The walls were very irregular. This cavity communicated by a narrow orifice with the smaller cavity which was more anterior. A rather large bronchial outlet was visualized in the lower part of the cavity opening on the tip of a nipplelike elevation of the wall. During inspiration air bubbled freely through this outlet. The same experiment with saline was tried as in Case 1, and showed that positive pressure, created by injection of air into the cavity, did not cause expulsion of it, whereas suction of the air of the cavity produced its immediate elimination.

Lipiodol and methylene blue were injected into the cavity; the dye was expectorated almost immediately, while on a film taken twenty minutes after injection, lipiodol was seen in the cavity and the draining bronchus was well visualized (Feb. 14, 1938, Fig. 13). Lipiodol was still present two days later (Feb. 16, 1938, Fig. 14). It had disappeared completely within four days. In a lateral film taken on March 11 no trace of lipiodol could be found (Fig. 15).

Fig. 9. Fig. 10.

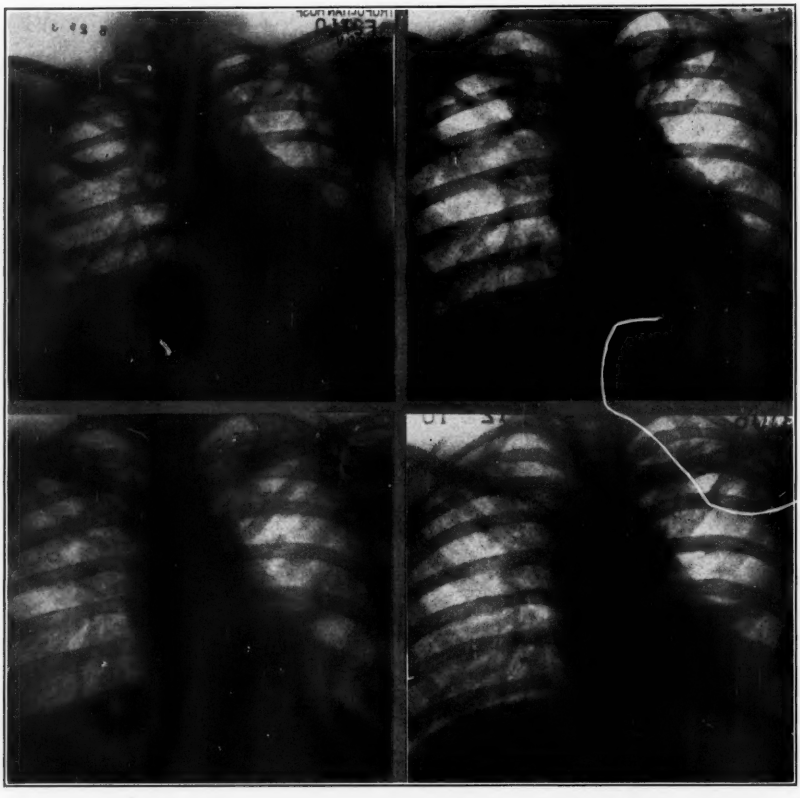

Fig. 11. Fig. 12.

Fig. 9.—Case 2. A white female with bilateral tuberculosis. Note the giant spherical cavity in the right upper lobe. Note the thick walls of the cavity and the fluid level. In the left lung there is a small anterior spherical cavity and an irregular, posterior, pear-shaped cavity with fluid level. (May 25, 1937.)

Fig. 10.—Case 2. Taken forty-three days later than the x-ray, Fig. 9. Both large cavities have increased in size. Note the changes in the density of the walls of all the cavities. (July 7, 1937.)

Fig. 11.—Case 2. Taken about three months later than the x-ray, Fig. 10. Note the change in size of the cavity in the right upper lobe.

Fig. 12.—Case 2. Taken two months later than the x-ray, Fig. 11. Note the complete spontaneous closure of the cavity of the right apex, whereas the cavities in the left lung have enlarged. The pear-shaped large posterior cavity is much enlarged and has a fluid level.

During quiet inspiration this cavity showed moderate increase in size. On the contrary no change could be seen during forcible expiration or during cough. This is easily explained by what had occurred to the saline injected into the cavity when the intracavitary pressure was increased. It is obvious that during forcible expiration the intracavitary pressure rose and caused closure of the bronchial outlet preventing

Fig. 13. Fig. 14.

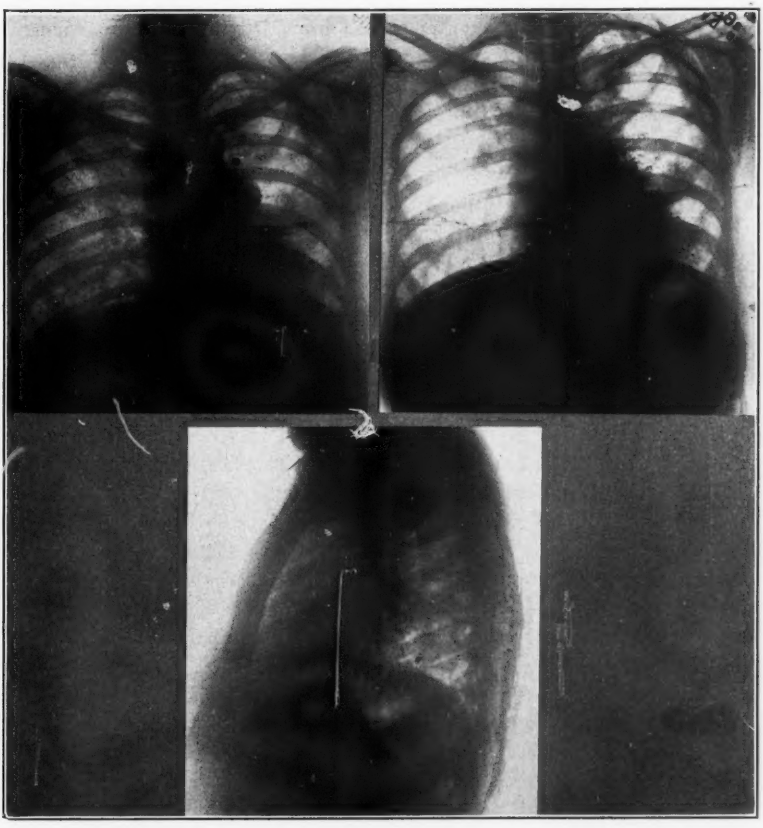

Fig. 15.

Fig. 13.—Case 2. After direct injection of lipiodol and methylene blue solution into the cavity. The dye was expectorated almost immediately. The lipiodol can be seen in the cavity in the draining bronchus and in the adjacent lung parenchyma. (Feb. 14, 1938.)

Fig. 14.—Case 2. Taken two days later than the x-ray, Fig. 13, showing lipiodol still present in the cavity, bronchi, and lung parenchyma.

Fig. 15.—Case 2. Taken twenty-three days later than the x-ray, Fig. 14. The lipiodol has been completely evacuated from the cavity and left lung. (March 11, 1938.)

any decrease of the size of the cavity. The explanation of closure of the bronchial outlet when the pressure in the cavity is raised is easy to

understand when we consider that the same phenomenon occurs in the distended urinary bladder in which partial aspiration of urine may relieve the valvelike closure at the bladder neck. In this patient, 1 c.c. of monolate solution was injected around the orifice of the draining bronchus with the intention of producing cicatricial closure of it. No untoward effects followed this procedure, but neither was the orifice closed. Occasionally the draining bronchus may be so wide that completely free ingress and egress of air takes place during respiration. We have observed only one such case in which marked expansion of the cavity during inspiration and retraction during expiration were present on cavernoscopic and fluoroscopic examination. The pathology of the draining bronchi explains the rarity of these cavities.

3. PATHOLOGIC FINDINGS

In advanced cases that came to autopsy, large communications between the bronchial tree and the cavities were frequently present. It is obvious that through these large holes air circulates freely into the cavities, and the latter may present respiratory expansion and retraction. However, usually tuberculous cavities communicate with the bronchial tree through narrow and long draining bronchi. Ameuille, Delhomme, and Raust[12] have published lately an excellent study of the pathology of these bronchi.

Draining bronchi were well known and described by the older phthisiologists; Laënnec, Louis, Andral, Grancher et Gruvelhier, in France, have given detailed description of them.

It is easy to understand the formation of these bronchi. When a patch of tuberculous pneumonia undergoes caseation and liquefaction the bronchi of the area of the parenchyma are destroyed at the same time and the liquefied caseum is emptied through these outlets. It is obvious that a cavity may communicate with several bronchi and also that the larger the destroyed area the larger is the diameter of the draining bronchus. When a whole lobe has sloughed out the draining bronchus of the cavity is necessarily represented by a lobar bronchus. This point is very important because the difficulty of closure of a large lobar bronchus explains the difficulty of closure of such large cavities. On the contrary, small peripheral cavities drained by small bronchi can close much more easily.

Older authors, as Letulle,[87] considered that bronchi can remain relatively healthy in a diseased portion of the lung. On the contrary Ameuille, Delhomme and Raust have shown that bronchi located in the diseased parenchyma present marked lesions of their mucosa, muscularis, glands, and cartilages; these lesions are more accentuated near the cavity and decrease in importance as the draining bronchus approaches the larger and healthy bronchi. The latter generally present slight lesions, if any.

Occasionally several bronchi may start from the cavity; however most frequently they converge to a larger outlet which represents the main draining bronchus. The orifice of this bronchus in the cavity is round or oval, level with the wall of the cavity, or it opens into the tip of a small elevation. It is not rare to find it in the bottom of a funnellike depression of the wall of the cavity. Generally this draining bronchus terminates into the cavity obliquely, like the mouthpiece of a saxophone (bec-de-flute, Ameuille). This explains the ease with which a valve-like action can develop at this point; this explains also the closure of this outlet when the intracavitary pressure increases, and its opening when pressure decreases. The mucosa of the draining bronchus is edematous, reddish, irregular and covered with mucopurulent exudate. The epithelium is desquamated and partly destroyed. The chorium presents marked dilation of its capillaries due to endarteritis and obliteration of larger bronchial branches which cause the development of collateral capillary circulation. The mucous glands present important changes; their excretory ducts are often found to be obstructed and their acini dilated. These glandular lesions enhance the irregularities and narrowing of the lumina of these bronchi and favor their temporary occlusion. The smooth fibers are dissociated and often completely destroyed and replaced by collagen and connective tissue. The cartilages are easily attacked by tuberculous caseation, and often only fragments of them can be seen. As the muscular tissue is destroyed, connective tissue is developed leading to fibrous peribronchitis, which is the most important factor in the permanent and definite closure of these bronchi and of healing of cavities. Thus the pathology of the draining bronchi explains the mechanism of their temporary and incomplete obstruction, either by inflammatory edema or by caseous or mucous plug or by blood clot, and also it accounts for the possibility of their reopening after temporary closure causing reopening of the cavity. Finally it gives a simple and clear explanation of the mechanism of complete and definite closure of the draining bronchus by fibrotic changes. Moreover, the pathology of the draining bronchus gives us, we believe, the key to the solution of a problem for which no solution has been proposed so far, namely, why giant cavities which have not closed with pneumothorax often do so following thoracoplasty. Our explanation is that thoracoplasty causes permanent collapse of the lung and permanent atelectasis. Coryllos has shown that under these conditions permanent ischemia of the atelectatic lung is produced, causing rapid proliferation of connective tissue. The same phenomenon is observed in the well-known ischemic paralysis of Volkmann, occurring after prolonged compression of the arm, whereas the extensive fibrotic changes of the muscles of the forearm observed in this syndrome do not occur if equally strong, but intermittent, compression was applied on the arm. The difference between thoracoplasty and pneumothorax is that the first causes

persistent ischemia and anoxemia of the collapsed lung, while in pneumothorax ischemia and collapse vary with the absorption of air and consequently are only intermittent.

The pathologic study of Ameuille, Delhomme, and Raust is well supplemented by the radiologic and pathologic investigation of Salkin, Cadden, and McIndoe. These authors have injected tuberculous cavities through the chest wall after death with radiopaque solutions, while the cadaver was fixed on a chair in erect position. They found that out of 142 cavities drainage was accomplished by several bronchi only in two (1.4 per cent), while in the remaining 140 (98.6 per cent) draining was insured by a single draining bronchus. This bronchus was located in the majority of cases at the most dependent part of the cavity in the upper lobes and in any part of the cavity, with equal frequency, in the lower lobes. Thus we must accept definitely that the draining of a cavity depends upon its main draining bronchus.

Of the 147 cavities injected by these authors the opaque solution was retained in only fourteen cavities (9.5 per cent). This proves the great predominance of cavities have an open draining bronchi (90.5 per cent). These authors gave to the cavities in which the opaque solution was retained the name of blocked cavities. Fifteen of these large blocked cavities had been sectioned in order to determine the nature of the block. It is interesting to note that in eleven (73.3 per cent) the block was due either to plug by mucous or caseous material or to inflammatory swelling. Only in two cases (13.3 per cent) obstruction of the bronchus was due to fibrosis. This point is very important because it explains the temporary and transient character of the block of these cavities, which is found in almost every one of the clinical cases reported by the same authors. More recently Eloesser[46] has also studied these blocked cavities on living patients. He arrived at the conclusion that the conception of Coryllcs was wrong; Eloesser states that "closure of the draining bronchus does not necessarily cause collapse of the cavity as Coryllos has supported." On the contrary Holcomb and Weber,[64] Korol,[81] Tchertkoff and Bobrowitz,[138] Fleischner, Palacio and Mazzei,[108] Trocmé,[142] Bernou and Vivant,[19] Jaquerod[76] (1936), Kapuszewski,[25] Luzzatto,[90] Naveau,[100] Jacobs and Beloff,[73] and almost all the authors who have studied this problem since 1936 have accepted the conception of Coryllos.

4. PATHOLOGIC PHYSIOLOGY OF GIANT CAVITIES

We are now in possession of the necessary data for a more detailed study of the pathologic physiology of giant cavities. It is obvious that what is true for giant cavities is also true for all tuberculous cavities, from which the former differ only in size.

All authors agree today that a patch of tuberculous pneumonia may follow two courses. Either the diseased area is healed by resolution or

it goes to caseation, according to the degree of allergic sensitivity of the pulmonary parenchyma and more especially to the dosage of the virulence of the infecting tubercle bacilli. The caseated area may become encapsulated and remain so permanently, often presenting progressive calcareous infiltration; or it is liquefied and the caseum is eliminated through the ulcerated bronchi, leaving a hole in the pulmonary parenchyma. This hole does not possess any real wall. A zone of reaction is formed early around the caseated area which is usually designated as the wall of the cavity. It should always be borne in mind, however, that in reality this zone belongs to the parenchyma of the lung and that its changes depend upon and are regulated by the changes of this parenchyma. It must be well understood that a cavity is not a cyst possessing its own wall but a mere hole in the lung, the wall of which is formed by the pulmonary parenchyma; many misunderstandings are due, we believe, to the erroneous conception of the "wall" of the cavity as being a part of the cavity.

If these fundamentals are well grasped the rest of the pathologic physiology, and mechanics of cavities can be easily understood provided that we strictly follow the general laws of physics and biology without allowing any deviation toward easier but unwarranted and rather metaphysical theories. In fact, a cavity being a hole in the lung communicating with the bronchial tree and the outside world through its draining bronchus, it contains air under atmospheric pressure. Since the rest of the lung is submitted to the sub-atmospheric intrapleural pressure, this cavity must necessarily assume a spherical shape;[118] if the normal elasticity of the lung is only little affected this hole naturally will appear in the x-ray larger than it is when the lung is extracted from the chest and the negative intrathoracic pressure is suppressed, causing collapse of the still elastic parenchyma; on the contrary there will be no changes in the size of the cavity if the surrounding parenchyma is atelectatic and fibrotic. On the other hand, tubercle bacilli will grow freely in the cavity because of the presence in it of adequate amounts of nutrient material and especially of oxygen. Thus a tuberculous cavity represents a laboratory flask in which tubercle bacilli are growing; through the neck of this flask, represented by the draining bronchus, bacilli pass into the bronchial tree rendering the sputum positive. Concurrently tuberculin is produced freely and is absorbed by the tissues causing toxic symptoms and enhancing tissue sensitivity both general and local.

This being the case it is obvious that the future course of the cavity will be regulated by the fundamental laws of the pathologic physiology of the lung, and consequently it will depend greatly upon the changes which may occur in its draining bronchus.

Three eventualities may occur. The first eventuality is that this bronchus remains patent (Fig. 16, I). In that case growth of tubercle

bacilli will continue unhampered, bronchogenic spread of the disease will be unchecked, toxic phenomena will be present, and the cavity will gradually increase by progresive sloughing of the lung. Because of constant production and absorption of tuberculin, perifocal infiltration and exudative thickening of the wall will take place. In other words we deal in these cases with an active lesion and an open cavity, and this term of "open lesion" finds here its anatomic, pathologic and biologic justification.

The second eventuality is that the bronchus may close (Fig. 16, III). If this closure is complete the air entrapped in the cavity is bound to be absorbed. This always occurs wherever in a living organism air becomes entrapped, be it in the subcutaneous tissue, the peritoneum, the ventricles of the brain, and even in the pleural cavity and obviously more so in the lung, in which gas exchanges are more active and in which bronchial obstruction leads necessarily to atelectasis. It is obvious

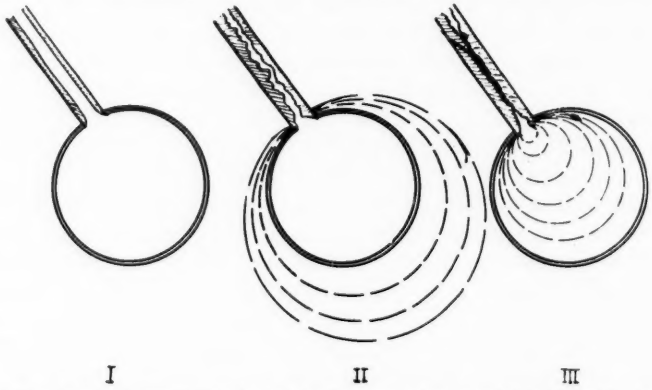

Fig. 16.—Schematic representation of changes occurring in cavities following changes in the patency of their draining bronchus. I, open bronchus; II, narrowed bronchus (tension or check valve cavity); III, closed bronchus; cavity disappearing.

that the results of this complete closure will be threefold: The first is a mechanical effect, namely, severance of communication of the cavity with the bronchial tree, hence the sputum turns negative for tubercle bacilli. The second effect is physiologic, namely, atelectatic shrinkage and closure of the cavity. The third effect is biologic, namely absorption of oxygen and creation of unfavorable conditions for growth of tubercle bacilli. Atelectatic shrinkage and concentric closure of cavities is easily explained by the absorption of the gases contained in the cavity. In fact Henderson and Henderson have shown that under the conditions realized by the obstruction of the draining bronchus, a negative pressure of –56 cm. of Hg may develop in the cavity, that is, equal to the difference between the partial tensions of gases in the atmospheric air and the venous air; this tremendous difference in intra- and extracavitary pressures is sufficient to force almost any cavity to

contract concentrically and to close; concurrently, the surrounding parenchyma expands, becomes emphysematous and fills the space liberated by the shrinkage of the cavity. The speed of closure of the cavity depends especially and predominantly upon the completeness of the obstruction of the draining bronchus and secondarily upon the activity of the circulation and gas exchanges around the cavity. However, even very slow circulation in the surrounding parenchyma is sufficient to insure complete absorption of gases and especially of the oxygen entrapped into the cavity, as it was shown by Coryllos and Birnbaum.[32, 33, 36, 37] Therefore, occasionally cavities do disappear with disconcerting rapidity; this mechanism explains also that at the place of the closed cavity no scar can be found either in the radiograms or even at the autopsy table, although a linear scar left by the closed draining bronchus is often visible in the radiograms, as Tchertkoff and Bobrowitz[138] have shown. We shall take up this point later.

Besides the mechanical and physiologic effects of closure of the draining bronchus, there is a biologic effect. This is due to the rapid absorption of the oxygen of the air entrapped in the cavity and the impossibility of its renewal. As Coryllos[32] has shown, the degree of anoxemia developed in the cavities following closure of the bronchus is extreme and amply sufficient to cause arrest of the growth of tubercle bacilli. Pinner[110] objects to this point and doubts whether the degree of anoxemia produced in the cavity is sufficient to be bactericidal. But there is no question of anoxemia causing death of the bacilli but only causing arrest of their growth and of their metabolic processes. This can be and is accomplished by the degree of anoxemia developed in the closed cavities (less than 0.01 per cent) as Novy and Soule[103] on one hand and Loebel, Shorr, and Richardson,[88] on the other, have proved experimentally. With the arrest of the growth of tubercle bacilli and of the production of tuberculin, toxic phenomena and tissue sensitivity decrease. A corollary of this conception is a fact of paramount importance which, to our knowledge, had never been mentioned before. This is that the thickness of the wall of the cavity and the degree of perifocal infiltration of the parenchyma surrounding the cavity are associated with the patency of the bronchus of the cavity. This offers for the first time a rational explanation of the peculiar changes in the thickness of the walls of these cavities, which could not be understood with the generally accepted opinion that the thickness of the wall was due to fibrotic changes which are irreversible. A number of cases reported by Ornstein and Ulmar[106] in their article on cavities in Goldberg's textbook offer an impressive illustration of this phenomenon. This leads to the important conclusion that "the thickness of the wall depends upon the patency of the draining bronchus."

At this point we want to present another factor as evidence in favor of the conception of Coryllos. This is the striking similarity between

a tuberculous cavity and a tuberculous empyema cavity with bronchial fistula. So long as the draining bronchus of a pulmonary cavity is open, pressure in the cavity is atmospheric, or above atmospheric when there is valve action of its bronchus; the oxygen content of the cavity is high, tubercle bacilli grow freely, sputum is positive, and danger of spread of the infection to the healthy lung is present, and the cavity necessarily remains open and visible in the x-ray films. Closure of the bronchus on the contrary causes absorption of gases, production of negative pressure in the cavity, expansion of the surrounding parenchyma, and disappearance of the cavity, hence, negative sputum, arrest of growth of tubercle bacilli, and decrease of toxicity because of decreased production of tuberculin. Likewise, in tuberculous empyema with bronchial fistula, as long as the bronchial fistula is patent, pressure in the empyema cavity is equal or above atmospheric, if a valve action is present in the bronchial fistula (tension pneumothorax corresponding to tension cavity); the empyema cavity cannot close, oxygen content is high, consequently, bacilli grow freely, sputum is positive, and danger of spread of the infection to the healthy lung is present. On the contrary, following closure of the bronchial fistula the pressure in the empyema cavity becomes negative, gases, especially oxygen, are rapidly absorbed, the surrounding lung is forced to expand, and the empyema cavity decreases and vanishes while the sputum becomes negative and toxic phenomena disappear. This likeness of tuberculous cavity and empyema extends further; in fact, as absence of expectoration of dye or lipiodol introduced into the empyema cavity does not necessarily mean absence or complete closure of bronchial fistula, likewise, nonexpectoration of dye or lipiodol injected into a tuberculous cavity does not necessarily mean complete closure of its draining bronchus. Likewise tension pneumothorax is homologous to tension cavities and in both the bronchial outlets are valvelike but open.

It is consistent with the conception of Coryllos that closure of the cavity will persist as long as its draining bronchus remains closed. Reopening of the bronchus will lead to the re-expansion of the cavity which, thus, will reappear at the same place where it was before. Thus the mysterious, baffling, and inexplicable appearance and disappearance of cavities, for which so many fantastic theories have been invented, finds a simple explanation based on fundamental physical laws.

The third eventuality which may occur in the draining bronchus is its narrowing to a degree allowing ingress of air during inspiratory dilation of the draining bronchus but forbidding its egress during the expiratory narrowing of the draining bronchus (Fig. 16, II). This again is in accordance with fundamental physical laws. It is the well-known mechanism of obstructive empyema which is due to partial bronchial obstruction whatever may be its cause, foreign body, intrabronchial tumors, extrabronchial compressions, and even narrowing

of bronchioli, as in allergic emphysema. In the latter, the typical picture of which is realized in the allergic shock of the guinea pig, the narrowing of the small bronchioli which is caused by the spastic contraction of their circular fibers or muscles of Reisseissen is the cause of the characteristic and marked emphysema of the lung. Chevalier Jackson and Manges have exhaustively studied this phenomenon of obstructive emphysema.

Thus the conception of Coryllos leaves no room, we believe, for any doubt and renders unnecessary any involved theories and further discussions. During the past four years and more especially during the last two years, Coryllos' conception has been accepted almost unanimously, except for a few dissenting opponents (Pinner, Salkin et al. and Eloesser).

It is clear that intermittent and incomplete closure of the draining bronchi cannot produce any marked and permanent changes in the cavity. In this category should be placed the so-called block cavities of Salkin, Cadden, and McIndoe, and of Eloesser. In fact, as has already been stated, the terms blocked cavity and blocked bronchus have no physiologic meaning whatever, so long as they do not give any precise definition of the only important factor in the evolution of cavities, namely, whether these bronchi are really open or closed.

It is our contention that in the so-called blocked cavities the bronchi are open. We base this contention upon the following evidence, drawn not only from our observations but also from the careful study of the very papers of the authors who disclaim the theory of Coryllos.

First, the pressure in the cavity of the cases of Eloesser was positive. This would be impossible if the draining bronchus were closed. Second, aspiration of air from the cavity did not change the size of these cavities, which should occur if the bronchi were closed. Third, when a catheter was introduced into these cavities and suction was applied, air could be aspirated freely without producing collapse of the cavity. In one of his cases Eloesser aspirated air for several hours with a Connell suction machine. The result he obtained was, contrary to his expectation, that not only the cavity did not collapse, but on the contrary, the draining bronchus which was found blocked before aspiration, as indicated by the retention of lipiodol in the cavity previously injected into it, did open, and lipiodol passed into the bronchial tree. It is obvious that the air aspirated by the suction machine was the air of the room passing into the cavity through the bronchial tree. Of course this could not happen if the draining bronchus of the cavity was closed. Fourth, because in every one of the clinical cases of Salkin, Cadden, and McIndoe and of Eloesser the draining bronchus was alternatively open and closed; sometime in the course of the disease the cavity was filled up with fluid, and the air disappeared (closed bronchus), and sometimes air and fluid level were present. Most characteristic is Case 3 of Salkin, Cadden, and

McIndoe. "In March, 1933," write these authors, "the cavity appeared as a dense shadow and the sputum was negative. In May, 1933, the cavity reappeared and sputum became positive. In February, 1934, the cavity was again replaced by a dense shadow and the sputum was negative. In June, 1934, the cavity reappeared and sputum became positive. In October, 1935, the cavity was again replaced by the dense shadow with negative sputum." Thus we have here a typical example of intermittent closure and opening of the draining bronchus, which is identical to the case of Morin and Waitz quoted above. This is proved beyond any doubt by the constant coincidence of filling up of the cavity and of negative sputum. However, it is upon these cases that these authors[123] have based the statement that "their study refutes the belief of Coryllos that closing of a draining bronchus causes healing of a cavity."

Likewise Eloesser[46] came to the conclusion "that total and permanent obstruction (of draining bronchus), if there is such a thing, is a factor that causes cavities to resist attempts at closure." This is a very sweeping statement. It is therefore necessary to study more closely the cases upon which this statement was based and to find out whether Eloesser or Coryllos is right in their diametrically opposite conceptions.

At the onset one is impressed at the perusal of the case histories of Eloesser[46] by the fact that the author himself gives ample proof that in none of his nine cases was there a permanent closure of the draining bronchus.

In Case 2, of this author, the cavity was needled and air was aspirated. Lipiodol and methylene blue were injected, which were not expectorated. But four days later the cavity was needled again and this time cough had caused elimination of the dye. Eloesser admits that the previously blocked bronchial communication was now open. "It seems," he says, "as though the bronchial opening was only temporarily closed." A catheter was then introduced into the cavity through a trocar; immediately the bronchial block was released and the patient began to expectorate blood and lipiodol "which she had never done before." No reduction in the size of the cavity took place although aspiration of large amounts of air was done. On the contrary injection of a small amount of air through the catheter increased the size of the cavity. Eloesser himself admits that "a valve mechanism was therefore apparent." This patient had later two stages of thoracoplasty which had produced closure of the cavity and conversion of sputum. Therefore, in this case blocking of bronchus did not close the cavity whereas thoracoplasty caused closure and disappearance of the cavity. Why? There is we believe the crux of the whole question; Eloesser thinks that there is no answer to this question. We have already shown that there is an answer and that this answer is very simple, if we accept the conception of Coryllos.

In Case 3 of the same author needling of a large apical cavity disclosed pressures $+1\frac{1}{2}$ $-1\frac{1}{2}$, that is, the same as in the bronchi. Lipiodol was not expectorated. However, twelve days after the second-stage thoracoplasty the cavity measured only 3 cm. in diameter and was rapidly closing. Again, how can atmospheric pressure in the cavity be explained if the draining bronchus was closed?

Case 4, of Eloesser, concerns a patient with a large thin-walled cavity which did not collapse by pneumothorax. However, following first-stage thoracoplasty with apicolysis "the cavity was seen to blow in and out with respiration"; Eloesser concluded that "after apicolysis the bronchus seemed to open." A week later the cavity was displaced downwards by the operation, but its size did not change. Three days later the cavity was needled and pressure was found –4 +3. Forced expiration blew the water out of the manometer at a reading of more than +12. This can leave no doubt that the bronchus was open, although the dye injected in this cavity was expectorated only nine hours later. As in the previous case a catheter was introduced into the cavity through the chest wall and "suction drainage was instituted with the hope to collapse it." Several ounces of pus swarming with tubercle bacilli were withdrawn, which showed that the bronchus again was closed. The catheter was then connected with a Connell suction apparatus. The cavity was not reduced in size; on the contrary "suction opened the bronchial communication making further attempt at suction futile." The patient wore this tube for months without any change in the cavity. But again subsequent thoracoplasty has closed this cavity.

Similar phenomena were observed in every one of the nine cases of this author. Atmospheric or positive pressures were present in the cavities; alternating closure and opening of the draining bronchi were noted in every case; opening of temporarily closed bronchi was caused by suction, which failed in every case to decrease the size of the cavity although hundreds of cubic feet of air were aspirated from them. This evidence proves beyond any possible doubt, we believe, that in none of the cases of this author was there "a permanent and complete closure of the bronchus." Therefore the statement of this author that "total and permanent closure of draining bronchus does not produce closure of the cavity" not only is not supported by his own cases but on the contrary it is refuted by the evidence that he has presented.

The fact that thoracoplasty succeeded in closing the cavities in which aspiration failed to do so is obviously the result of the fact that the former caused definite and total closure of the bronchus by the mechanism explained previously, whereas the second failed to do so, unless Eloesser is willing to admit that a cavity can close while its draining bronchus remains open. On the other hand, our cavernoscope findings offer a very simple explanation of the phenomena observed by Eloesser

and corroborated our opinion that in all these blocked cavities the bronchi were open at least intermittently.

Our explanation of the mechanics of the intermittent closure of the draining bronchus is as follows: During inspiration and especially during deep inspiratory movements preceding cough, which decrease the pressure in the cavity as in the rest of the lung, air is sucked into the cavity and pressure in it becomes positive. As long as pressure remains positive the intracavitary bronchial outlet is compressed and closed. But gradually the entrapped air, especially the oxygen, is absorbed and pressure decreases. As pressure decreases new air penetrates through the bronchus. This may be accomplished very slowly so that lipiodol or even dye injected into the cavity may remain in it. On the contrary, when the intracavitary pressure is rapidly decreased, as when Eloesser introduced a catheter in it and more so when he applied vigorous aspiration, the valve action of the bronchial outlet is released and dye and lipiodol are evacuated into the bronchial tree. This conception is based upon our cavernoscopic findings and is in agreement with fundamental physical laws. Moreover, the same phenomena occur in all elastic cavities containing fluid or air under pressure and connected with narrow outlets. Tension pneumothorax and overfilled urinary bladder are suitable examples.

A last point to be discussed is why a number of giant cavities expand and retract during respiration while others remain unchanged. We believe that here also the condition of the draining bronchus is responsible for these differences. In fact when the cavity communicates with the bronchial tree through a wide open bronchus or through a hole, air enters the cavity freely during inspiration and is expelled from it during expiration. Therefore, if the diseased lobe has remained sufficiently elastic as to expand during inspiration and retract during expiration, the cavity will present the same changes. On the contrary in tension giant cavities, which communicate with the bronchial tree through a narrow draining bronchus, air enters into the cavity only during inspiration but it cannot be expelled during expiration. As positive pressure is gradually built in the cavity during inspiratory movements, the orifice of its draining bronchus is compressed and closed. Thus not only air cannot leave the cavity during expiration but it cannot enter into it even during inspiration when the pressure in the cavity has become equal or higher than in the bronchial tree. Consequently there can be no changes in the size of these cavities during respiration.

During cough changes in size of giant cavities are again regulated by the condition of the draining bronchus. These changes should be considered in relation to each of the three phases of cough. Coryllos[34] has shown that cough is not a simple expiratory phenomenon but it is composed of three phases: one inspiratory, one compressive, and one expulsive. Cavities with open bronchus expand during the first or in-

spiratory phase. They remain unchanged during the compressive phase, during which the glottis is closed, and intrathoracic and intrapulmonary pressure are uniformally increased so that intrabronchial pressure is equal to the pressure in the cavity. On the contrary during the third or expulsive phase, during which the glottis opens and the compressed air is forcibly expelled, pressure in the lung falls, the lung retracts, and the cavity with widely open bronchus does the same. A tension cavity behaves differently; during the inspiratory phase the cavity cannot expand if the positive pressure in it is already higher than or equal to the negative intrapulmonary pressure created by the expansion of the chest; during the compressive period no change can occur in the tension cavity because the pressure in it is further increased so that the

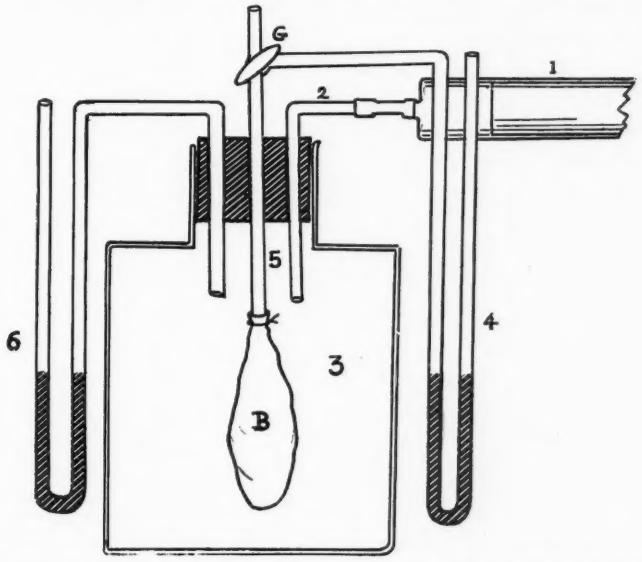

Fig. 17.—Apparatus of Ornstein and Ulmar explaining changes in the size of the cavity during respiration and cough (see text).

orifice of the draining bronchus remains closed; during the expulsive phase, on the contrary, tension cavities may show increase in size. This may seem paradoxical at first sight. Really it is what should be expected. In fact, during the expulsive phase intrapulmonary pressures fall suddenly because of the opening of the glottis and the rapid expulsion of the intrapulmonary air; but the air contained in tension cavities cannot be expelled; therefore, at this phase the pressure in the cavity may be higher than in the lung and consequently a large tension cavity with elastic walls may show a marked expansion at the end of cough.

An experimental study of the changes of pressures in giant cavities was made by Ornstein and Ulmar by means of a simple apparatus. A

large bottle was used, the neck of which was closed by a stopcock through which three glass tubes were passed (Fig. 17). Through tube 2 air could be pumped into the bottle or aspirated from it by means of a hand pump (1) in this manner causing changes of the pressure in the bottle. Tube 6 is a mercury manometer recording changes of pressure in the bottle. Tube 5 could be connected either with a mercury manometer (*4*) or with the outside air by means of a two-way stopcock (*G*). At the end of this tube was attached a rubber balloon (*B*) representing the cavity. Thus the bottle represents the chest in which pressures varied from negative (inspiration) to positive (expiration). When the stopcock (*G*) was turned so that the cavity was connected with the outside air, an increase of pressure in the bottle (expiration) caused collapse of the balloon while decrease of pressure (inspiration) caused expansion of the balloon. It is exactly what happens in giant cavities which have large and widely open draining bronchi. On the contrary when the stopcock (*G*) was turned to the other direction so that communication of the balloon with the outside air was suppressed and connection with manometer (*4*) was established, changes of pressure in the bottle had little effect upon the size of the cavity. This is what occurs in tension cavities. But when a strongly positive pressure established in the bottle and it was suddenly released, the balloon expanded because the pressure in the balloon was higher than in the bottle at the time of release of pressure; this is what occurs in tension cavities at the end of cough, provided that the diseased lung parenchyma and the wall of the cavity have conserved a sufficient degree of elasticity. When a flat rubber valve was attached to the end of tube 5, inside of the balloon (B), and the other end of the tube was connected with the outside air, the balloon expanded gradually and progressively at each decrease of the pressure in the bottle (inspiration) but did not retract when the pressure was increased (expiration). Thus gradually the balloon was inflated, reached a certain degree of inflation depending upon the value of depression in the bottles, and it remained stationary. At this time the pressure in the balloon was positive and equal but of opposite sign of the maximal negative pressure created in the bottle during suction of air (inspiration). This gives a clear explanation of what happens in expanding tension cavities under pneumothorax.

We have endeavored to present here the results of our study of the pathologic physiology of giant cavities. Although this study is far from being complete, it shows, we believe, the importance of the study of pathologic physiology of tuberculous cavities in the correct interpretation of phenomena observed in the cavities during the course of pulmonary tuberculosis. Moreover, it is of a great aid in the understanding of the mechanism of their spontaneous or therapeutic closure and in the selection of procedures aiming at their obliteration and cure.

5. HEALING OF GIANT CAVITIES

The evidence presented above, has proved, we believe, our contention that giant cavities are difficult to collapse because of the valvelike mechanism in the tension cavities and the wide open draining bronchus in the open cavities.

We shall not enter into Coryllos' discussion upon the different theories of closure of cavities. This point has been discussed elsewhere.[32]

We shall quote here a small number of recent papers which show the trend of actual opinions on this question.

Trocmé[142] in a recent paper (1936) presented a discussion of the different theories regarding the mechanism of closure of cavities. "If there is a case," says this author (p. 122), "where nothing can be explained without atelectasis this is the closure and disappearance of tuberculous cavities." Previous theories may be divided on closure by fibrotic retraction of pericavitary connective tissue (Pinner); but if this were true we should have gradual shrinkage of the cavity under the action of concentric peripheral fibers and crumpling of the internal fibers. In other words, we should always find a scar in the place of the healed cavity and this scar should show a peripheral layer of well-marked circular fibers with a crumpled lumen, that is, something similar to a section of the pylorus. But there are no such scars found in the place of closed cavities and as a rule there are no scars at all. Gräff,[60] De la Camp,[24] Dahlstedt,[41] Giegler,[56] Hübschmann,[65] Letulle,[87] and Roubier[120] have declared that they could not find any scar. Exceptional are the cases in which scars were found (two cases of Pinner,[110] two cases of Gilbert[57]); in all these four reported cases there was hyaline and not fibrotic scar tissue. On the other hand, if this theory of fibrotic retraction of cavities was true, closure should always be gradual and never sudden, and reopening should never occur. Moreover, old cavities with thick fibrotic walls should be the ones that should more readily close, whereas, paradoxically, the cavities with thin and elastic walls are the ones that generally close.

Because of the inability to explain closure of cavities by this mechanism, says Trocmé,[142] a number of authors declared that cavities which closed were not true cavities but false cavities (Sampson,[124] Amberson,[5] and Dahlstedt[41]). However pathologic studies did not uphold these conceptions.

Numerous other theories have been presented, such as closure by intracavitary proliferation (Baum,[16] Roubier and Doubrow[122]), which was denied by Pinner.[110] Other authors (Dumarest, Joly,[77] L. Bernard, Le Seve[86]) have attributed closure of cavities to centripetal compression by pericavitary emphysema; but it cannot be understood how emphysematous lung can compress diseased and hard tuberculous lung nor how soft emphysematous lung can crowd and compress tuberculous parenchyma while intrapleural pressure remains negative; it is well

known that even pneumothorax with positive pressure, effusion, or even oleothorax is often unable to collapse these cavities. Moreover, it has been already shown that this pericavitary emphysema is not the cause but the result of closure of cavities. Last but not least this theory is contradicted by cavities which often having resisted to pneumothorax were later closed spontaneously in the reexpanded lung.

Bernard, Harrenschmidt, and Morin[17] have supported the hypothesis that changes in size and eventually closure of cavities are due to the elasticity of the lung and more especially to a "reflex active contraction of the lung parenchyma caused by the lesion acting as an irritative cause." We are again here in the realm of metaphysics with a "lobe actively retracting in mass and choking the cavity." No explanation of the afferent and efferent legs and of the nervous center of this hypothetical reflex is given by these authors. In the discussion that followed this presentation Rist refused to accept it.

Contrary to these hypotheses is the theory that obstruction of the draining bronchus with atelectatic closure of the cavity would, according to Trocmé,[141] cause a centripetal shrinkage of the hole of the cavity. The connective tissue around it is very scanty, and no scar is visible; hardly should we find a few starlike intersected fibers; and this is precisely what was described in the specimen as remnants of closed cavities by the old pathologists and more recently by Pagel,[107] Pinner,[110] and others.

6. BACTERIOLOGIC FINDINGS

In two papers published in 1933, Faragó[49] had reported that anaerobic bacilli were isolated from the content of tuberculous cavities taken aseptically from the cadavers a few hours after death. This author had found *B. welchii* and other anaerobes in 100 per cent of all the thirty cases examined. The same investigation carried out by this author on the sputum of living patients with tuberculous cavities[49] disclosed the presence of anaerobes only in 12 per cent of the cases. Forty-two per cent of the Welch bacilli isolated from the cavities were very virulent and killed guinea pigs in which the cultures were inoculated within twenty-four hours.

These findings are of great importance and would justify the objections of Max Pinner[110] and of Eloesser[46] to the theory of Coryllos,[32] namely that closure of the draining bronchus and creation of anaerobic conditions in the cavity should favor the development of anaerobic organisms and would certainly produce serious suppurative complications of the pulmonary parenchyma accompanied by toxic symptoms.

Yet no such complications have been observed either by these authors or by us in cases in which retention of exudate was present and had persisted for a long time in the cavity. When we consider that a cavity measuring 5 cm. in diameter has a capacity of 125 c.c. and a cavity of 10 cm. has a capacity of 1,000 c.c., it is surprising that marked toxic

symptoms do not appear following retention of such amounts of pus, if this pus were infected with virulent microbes and especially with anaerobes, and when favorable conditions of anaerobism were realized as in these badly drained cavities. If these complications did exist they should certainly have been noticed in all cases of cavities with fluid level, and more especially in the blocked cavities of Salkin, Cadder, and McIndoe[122] and of Eloesser.[46] They should have been mentioned by all authors for certainly they would represent a rather common complication of cavities. In reality not only is there complete silence in the literature on such complications but, moreover, the majority of the authors consider the presence of a fluid level in the cavity as a favorable sign often preceding its closure.

This work of Faragó which was based on a good bacteriologic technique raises two questions. First, why was there this discrepancy between the incidence of *B. welchii* in the cavities after death (100 per cent) and in the sputum of living patients (12 per cent), and second, whether or not anaerobes were present in the cavities while these patients were still alive. In fact, patients who died with advanced pulmonary tuberculosis, which is generally complicated by intestinal tuberculosis, belong to the variety of patients dead from ulceronecrotic diseases. In the latter, Rassfeld[116] (1921) has found anaerobic organisms in cultures of the blood taken from the heart in 100 per cent of all cases used for his investigation. Moreover, Zeissler[146] (1928) has shown that in these cases defect of the mucosa either in the lung or elsewhere acts as a portal for a secondary, post-mortem anaerobic contamination and that through this portal *B. welchii* among other bacteria get into the blood stream.

On the other hand if *B. welchii* were present in 100 per cent of the cavities during life why were these bacilli not found in more than 12 per cent of the sputum examined, although bronchiectasis is a frequent complication in pulmonary tuberculosis. Therefore, we believe that the conclusions of Faragó are in discrepancy with clinical findings, and the fact that anaerobes were found in 100 per cent of cavities is strongly in favor of post-mortem contamination of the cavities.

However, even if we exclude anaerobes, the prevailing opinion at the present is that pyogenic micro-organisms are always present in the pus contained in these cavities. Ameuille[9] believed that impaired drainage of cavities was detrimental to the health of tuberculous patients and Pinner and Eloesser objected to the conception of Coryllos on the same grounds. It was therefore of interest to further investigate this problem on the living patients. For this purpose pus was taken from the cavity either by aseptic aspiration, by means of a needle introduced into the cavity through the chest wall, or during reoperation for uncollapsed cavities by previous thoracoplasty. During these operations, reported below, the cavities were opened with strict aseptic precautions. The specimens were taken in the operating room personally by the

director of our bacteriologic laboratory, Dr. S. A. Petroff, and were submitted to a thorough study which was carried out by himself. We had thus assembled all the necessary requirements for the solution of the problem. Complete elimination of every possibility of accidental contamination of the specimen and flawless bacteriologic technique was carried out. The results were really amazing. In all specimens of seven patients which were examined no other organisms were found outside of myriads of acid-fast bacilli. Not only were anaerobes absent but even ordinary pyogenic organisms such as staphylococcus or streptococcus could not be found either in cultures or by animal inoculations. The same negative findings were obtained by Dr. Bedall, director of the laboratory of the Veterans' Hospital in Castle Point, N. Y.

These findings put an end to the common belief that the tuberculous cavities present mixed infection and that pyogenic organisms are always present with tubercle bacilli. This belief unchecked and unchallenged has passed from author to author and from textbook to textbook. Our findings throw a new light on the absence of any toxic phenomena due to retention of fluid in the cavity. Thus, clinically, a tuberculous cavity can be compared to a pure tuberculous empyema or cold abscess and not to a mixed infection tuberculous empyema or to a secondarily infected tuberculous cold abscess. These findings represent cold facts, the checking of which is not difficult. Therefore as long as they are not disproved, the objections of Max Pinner and of Eloesser to the theory of Coryllos based upon this belief are of no value.

These bacteriologic findings are of considerable practical importance. In fact if the danger of obliteration of the draining bronchus is removed there is no reason why we should not try to obtain it surgically, especially in cavities in which all other means of collapse, including thoracoplasty, have failed. This has been the working hypothesis in our endeavors to obtain healing of these cavities surgically.

7. MANAGEMENT OF GIANT CAVITIES

A number of these cavities close following pneumothorax or thoraco plasty. Others, on the contrary, remain open. Therefore, we consider that these two procedures should be applied first, at least at the present time. Crushing of the phrenic nerve or exeresis did not give to us nor to Huriau[68] (six cases of giant cavities) and to other authors any satisfactory results. Closed pneumonolysis on the contrary when possible has given a number of satisfactory closures, although in others had failed or even caused greater expansion of the cavity.

Unfortunately, it is not possible to know in advance whether a giant cavity will close or not under pneumothorax. The behavior of the cavity may depend to some degree on the extensive adhesions of the cavity to the chest wall and on the thickness of its walls; we believe,

however, that the most important factor is the condition of its draining bronchus. The first two factors are of minor importance as compared with the third. In fact, cavities apparently fibrotic and adherent to the chest wall often respond very well to pneumothorax, and satisfactory selective collapse has been obtained or could be completed by pneumonolysis. Likewise very large giant cavities with very thick walls, and apparently hopeless, have responded very well to thoracoplasty. In these cases extensive apicolysis added to thoracoplasty has been a great help to us.

We believe therefore that the most important factor is the condition of the draining bronchus. We have been unable to obtain closure of cavities which presented pressures equal to atmospheric, expansion and retraction synchronous with respiratory movements on fluoroscopy, a composition of their gaseous content near to the atmospheric air, and which did not present a fluid level. These characteristics indicate a wide open draining bronchus of a rather large diameter. We deem it of interest to give here a short résumé of three such cases, one of which came to autopsy.

The first case was that of a white male (W. S.) (Fig. 18). There was a giant cavity occupying major part of the right upper lobe. Following pneumothorax the lower and middle lobes were collapsed while the cavity remained unchanged (Fig. 19). Three stages of thoracoplasty with apicolysis and two revisions were performed within six months without succeeding in rendering the sputum negative. In Fig. 20 the cavity remained visible, and a small fluid level could be seen in fluoroscopic examination. This patient died seven months later. At the autopsy a large cavity was found having replaced the upper lobe and communicating with the bronchial tree through the lobar bronchus. Contrary to this cavity which has remained open is the case of a female patient whose sputum became negative, and the cavity disappeared following a two-stage thoracoplasty. This patient died from other causes seven months after thoracoplasty. The cavity was collapsed and its draining bronchus was closed.

The second case was that of a white female, 23 (R. G.). A giant cavity was present in the upper left lobe occupying almost the whole lobe. Pressure was -3 +4; composition of its gaseous content was O_2 19.8 per cent, CO_2 0.2 per cent. No fluid level was present at any time. Pneumothorax could not be induced. Phrenicectomy on the left side had no effect upon the cavity (Fig. 21). First-stage thoracoplasty with extensive apicolysis was performed (Aug. 16, 1933). The cavity had dropped below the third space without any changes of its size (Fig. 22). Second stage was performed four weeks later. The cavity was decreased in size but was still present as a slit (Fig. 23). Sputum remained positive, and the patient died with extension of the disease.

Of still greater interest is the third case which shows that as long as the draining bronchus is open, not only does the cavity resist all attempts at collapse, but on the contrary it may expand under the collapsed chest wall.

This third case was that of a colored female (H. M.) (Fig. 24). A giant cavity with thick walls was present at the right apex. Pressures were -4 + 5. Analysis of

its gaseous content gave O_2 20.1 per cent. Pneumothorax could not be induced. First stage thoracoplasty with extensive apicolysis was performed in December, 1934, causing a shrinkage of the cavity (Fig. 25). Second and third stages were performed in January and February, 1935. Fig. 26 shows expansion of the cavity under the collapsed chest. This patient died with extension of the disease.

These three cases taken at random from a great number of similar cases show clearly the hopelessness of our present procedures.

Fig. 18. Fig. 19.

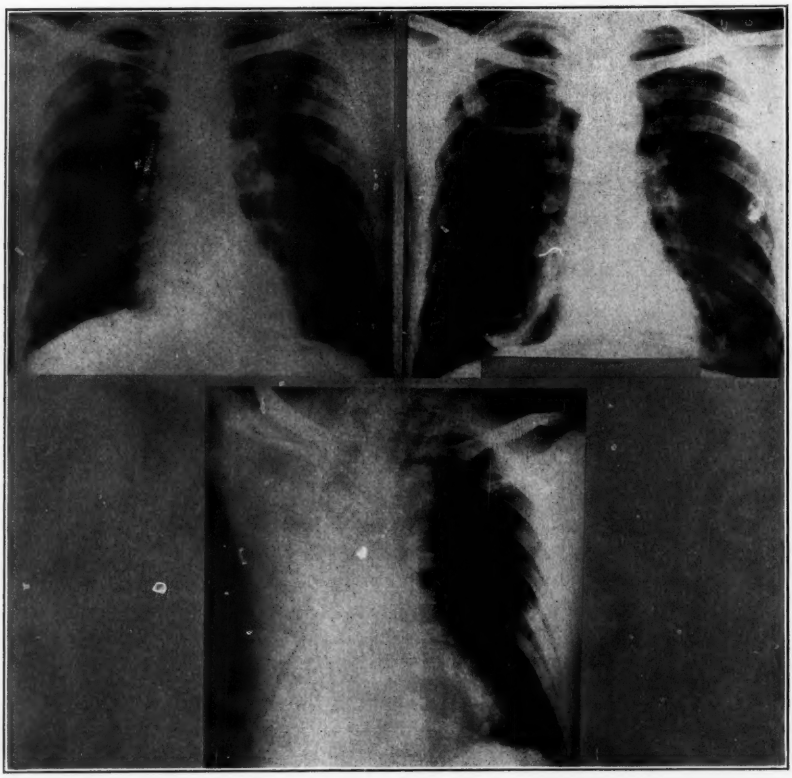

Fig. 20.

Fig. 18.—W. S. Giant cavity of right apex. June 6, 1932.

Fig. 19.—W. S. Oct. 31, 1933. Following pneumothorax the walls of the cavity have become thicker. Slight fluid level is present. Pneumothorax did not influence the cavity.

Fig. 20.—W. S. April 2, 1934. Three stages of thoracoplasty followed by two revisions did not collapse the cavity, although the whole skeleton of the right chest was removed.

We have been endeavoring for the past four years to close these cavities using different procedures with the hope of increasing the percentage, actually very low, of cure of these patients.

We have performed lobectomies in two cases. In one the upper lobe was removed. This was a colored female, upon whom three stages of thoracoplasty did not succeed in closing a giant open cavity. The upper

Fig. 21. Fig. 22.

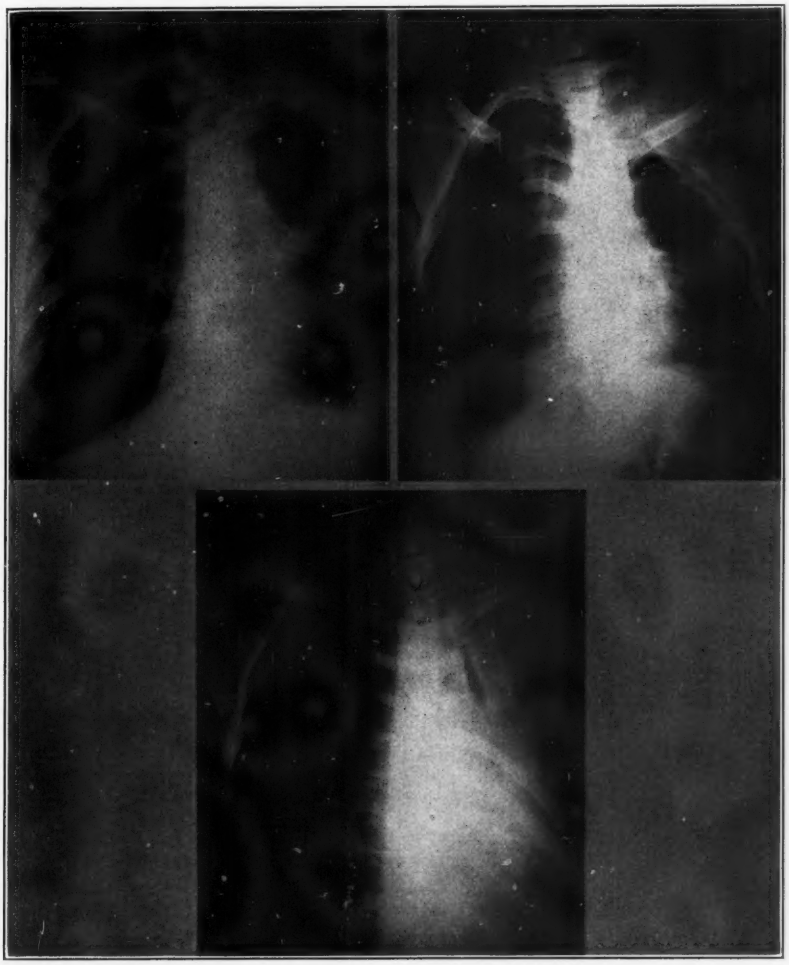

Fig. 23.

Fig. 21.—R. G. Giant cavity of the left apex, with open bronchus (Aug. 10, 1933).
Fig. 22.—R. G. First stage thoracoplasty produced marked drop of the cavity, without causing its closure (Aug. 19, 1933).
Fig. 23.—R. G. Second stage thoracoplasty has apparently decreased the size of the cavity; but it is still visible and sputum has remained positive. (Sept. 9, 1933.)

lobe could be separated from the parietal pleura and was removed. Careful closure of the bronchus was done. However a bronchial fistula

developed ten days after operation and the patient died five months later with extension of the disease. We had been unable to close the fistula.

The second patient, a white male, prsented two giant open cavities, one in the upper and the other in the lower right lobe. Pneumothorax

Fig. 24. Fig. 25.

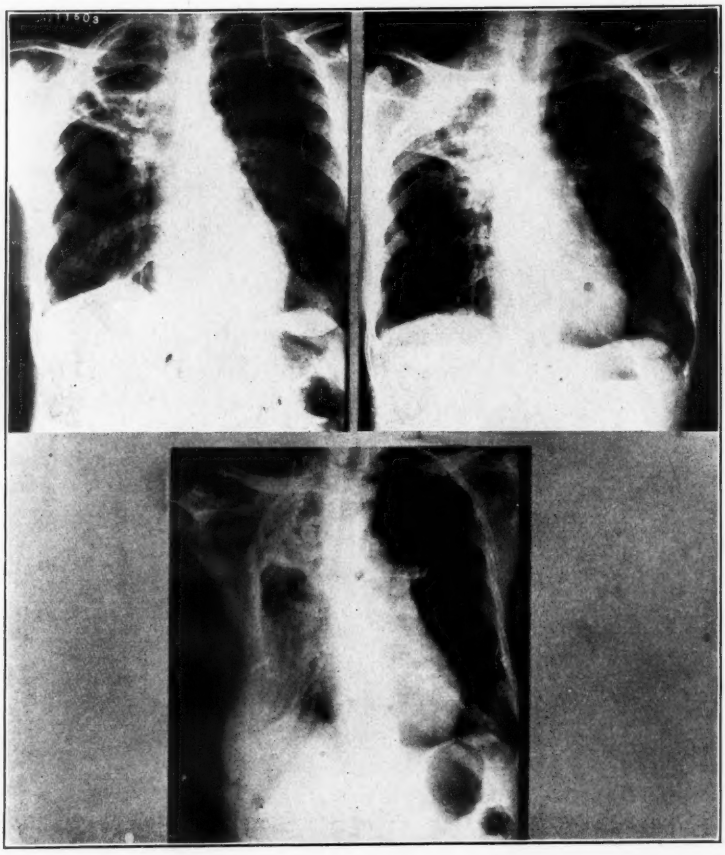

Fig. 26.

Fig. 24.—H. M. Giant cavity of the upper right lobe with open draining bronchus. (Oct. 31, 1934.)

Fig. 25.—H. M. Following first stage thoracoplasty, cavity has decreased in size. Sputum positive. (Jan. 19, 1935.)

Fig. 26.—H. M. Second and third stages of thoracoplasty did not succeed in closing the cavity; on the contrary the latter has "ballooned" under the collapsed area of the chest.

could not be induced. Three-stage thoracoplasty and phrenicectomy did not succeed in closing either of the cavities. During reoperation the lower lobe, which contained the larger cavity, could be separated

without great difficulty from the parietal pleura. The vessels of the pedicles of the lower and middle lobes were tied and sectioned; the bronchus was sectioned, carbolized, and sutured. Sputum became negative after operation. However a bronchial fistula appeared the sixteenth day. The patient died two months later with symptoms of amyloid nephrosis. The bronchial fistula had not closed up till the time of death, but sputum had remained negative.

Revisions of thoracoplasties with resection of the regenerated bone have given us 29 per cent good results in forty-three patients operated upon. This percentage cannot be considered as satisfactory. We have not been more fortunate with extensive anterolateral thoracoplasties.

However, we gathered an important lesson from our revisions. That is, when the apex could be stripped from the chest wall, lowered down, and anchored in this position, either by sutures or by placing above it a muscular flap at the place previously occupied by the apex, good results were obtained in over one-third of these cases (35.3 per cent). Unfortunately, it is not often possible to strip the uncollapsed cavity from the regenerated ribs. The uncollapsed cavities are usually much larger than it is usually suspected from the radiograms and their separation from the chest wall or of the lung from the parietal pleura is often impossible without tearing them.

Because of the unsatisfactory results obtained by the procedures enumerated above, and our conviction that closure of the draining bronchus should bring about healing of the cavity or at least render the sputum negative, we endeavored during the past years to attack directly the draining bronchus.

We did not attempt to close the lobar bronchus of the affected lobe because of the danger of suppurative pneumonitis of this lobe. In fact we have found that in the large bronchi of these patients pyogenic organisms are frequently present. However, Brooks[23] has lately reported a good result obtained by obstructing for six hours, by means of a rubber balloon, the lower right lobe which contained a large cavity. It should be noted, however, that in this case pneumothorax was present in the right pleural cavity.

We have attacked the draining bronchus by two different approaches. First, by injecting into the cavity, through the chest wall, different substances or by cauterizing the opening of the draining bronchus guided by our cavernoscope, under direct vision. The second procedure consisted in opening the cavity widely, during revision of thoracoplasty, and filling it with a large pedunculated muscular flap taken from the paravertebral muscles. We shall briefly report here the technique and results of these procedures which will be elaborated upon at a later date.

A. INTRACAVITARY, TRANSTHORACIC ATTEMPTS AT OBLITERATION OF DRAINING BRONCHUS

After accurate localization of the cavity a needle was introduced into it through the chest wall. When it was made certain that the needle was in the cavity, the following substances were injected into the cavity in six different cases. Solution of copper morrhuate; blood taken from the patient, simple or mixed with thrombin in order to produce rapid coagulation; silver nitrate (30 to 60 per cent solution); aseptic gelatine (8 per cent previously heated); and agar (1.5 per cent heated to 40° C.). We did not obtain any satisfactory results by these procedures. There were no untoward effects following these injections

In three other cases the cavernoscope was used. In the first we injected directly into the orifices of the draining bronchus a few drops of a 60 per cent solution of silver nitrate. We did not obtain closure of the bronchus. On the second case the mucosa of the draining bronchus was cauterized by means of a fine galvanocauteric knife introduced through the second cannula. This cauterization was carried out under the control of the cavernoscope as in closed pneumonolysis. Again we were not able to close this bronchus. In the third case we injected around the orifice of the draining bronchus, by the same technique, the sclerogenic solution used for injection of hernias (monolate). No closure of the bronchus was obtained.

B. INTRACAVITARY TRANSPLANTATION OF PEDUNCULATED MUSCULAR FLAP

The technique adopted for this procedure is as follows: An incision was made on the scar of previous thoracoplasty and the regenerated bony plate was excised. The parietal pleura was incised and the flaps dissected bluntly from the lung; by palpation the cavity was easily located in all cases and its presence was made certain by puncturing it; in all cases air and pus were aspirated; the cavity was then widely incised. In all our cavities there was blowing through the draining bronchus, although the orifice of the latter could not always be visualized. The size of these cavities varied from that of a large walnut to an orange. Methylene blue introduced into the cavity was expectorated in all our cases. The cavity was then carefully and gently sponged until it was perfectly dry. A long pedunculated muscular flap was raised from the paravertebral muscles and turned into the cavity so that it filled it completely. It was fixed in this position with interrupted stitches of catgut. The parietal pleura was closed above the flap and the wound closed in three layers. In four cases a small rubber dam was placed in the upper end of the wound. In eight cases the wound was closed without drainage after the results of bacteriologic

examination of their content, reported above, had shown that no pyogenic organisms were present in the purulent material contained in the cavities.

This procedure was carried out in twelve cases. The immediate results in all cases were very satisfactory. There was no mortality as a result of the operation, although the procedure was long and laborious, and all these cases were patients of long standing, presenting advanced amyloidosis, and on whom all other methods had failed to close the cavities and to convert the sputum.

In all patients except two, the wound had closed by primary healing. However, there was secondary opening between ten and twenty days after operation, and serohematic fluid was eliminated.

Of these twelve cases, six became negative immediately or shortly after the operation. One of these cases became positive again a month later.

Four of these twelve patients died. The first died three weeks later with acute bronchial obstruction, the second with uremia due to amyloid nephrosis; she had advanced amyloidosis with 100 per cent absorption of Congo red before this operation. The third patient, who had a bilateral case with pneumothorax on the other side, developed spontaneous contralateral pneumothorax and was deflated with marked relief. He died with bronchopneumonia in the contralateral side. The fourth was the best of the whole series. The patient became persistently negative two weeks after the operation and was ambulatory for four months. He contracted acute pneumococcic pneumonia in the contralateral lung and died.

In three of these cases autopsies were obtained and performed by Dr. Auerbach, the director of the laboratory of Sea View Hospital. The pathologic study of the specimens of patients who died from three weeks to four months after operation has disclosed a number of highly interesting points which can be summarized as follows.

During the first three weeks following operation an active tuberculization of the muscular flap took place. On the patient who died three weeks after operation the flap was in place, and no tuberculization could be seen (Fig. 27). It was adherent to the cavity by fibrin, but it could be easily detached. The patient who died four weeks after operation had an extensive tuberculization of the flap; caseated tubercles were found in it. The flap was filling the cavity and was adherent to its walls.

The autopsy of the patient who died four months after the operation disclosed a very interesting point. Not only the flap was in place but it was completely adherent to the walls of the cavity and it was transformed into fibrous tissue. The draining bronchus of the cavity was closed. The sputum of this patient became negative shortly after the operation and had remained such. Fig. 28 shows a frontal section of

Fig. 27.

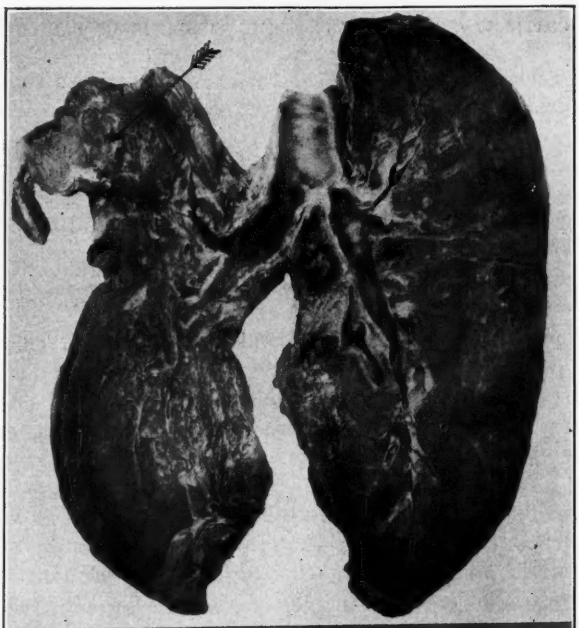

Fig. 28.

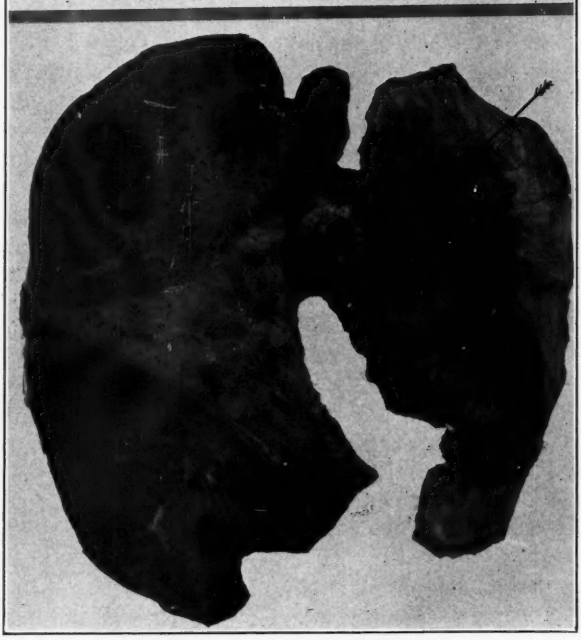

Fig. 27.—Specimen of the lung of patient operated by this procedure. Arrow shows the flap filling the cavity and adherent to its walls.

Fig. 28.—Specimen of the lung of patient operated by this procedure who four months after operation died of pneumococcic pneumonia of the other lung. Arrow shows perfect closure of cavity by muscular flap. This patient had persistently negative sputum following operation.

both lungs. The flap is well visible completely obliterating the cavity. The right lung is completely consolidated by pneumococcic lobar pneumonia. It is to be noted that this patient had positive sputum for four years after complete thoracoplasty and two revisions.

The lesson from these cases is that this procedure is an effective one. However, it is followed by tuberculization of the flap. If this could be prevented the complications reported could be avoided.

Fig. 29. Fig. 30.

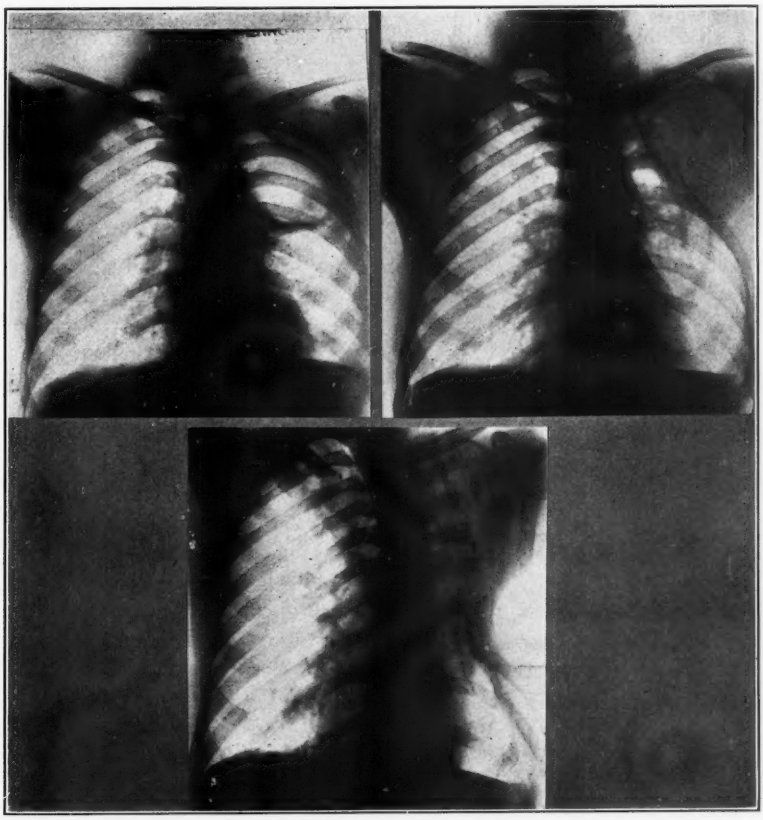

Fig. 31.

Fig. 29.—J. P. Tension cavity of the upper lobe. (May 10, 1937.)

Fig. 30.—J. P. Following two stages of thoracoplasty. Cavity still visible, but sputum was negative. (Aug. 31, 1937.)

Fig. 31.—J. P. Following third stage thoracoplasty. Sputum had become and remained persistently negative. Cavity is not visible in the radiogram. (Dec. 3, 1937.)

We can conclude that intracavitary injections of various chemical substances and of blood have been proved unsuccessful in our hands. Thoracoplasty with wide apicolysis is the procedure of choice when

pneumothorax and pneumonolysis have failed. Figs. 29, 30, and 31 show a successful closure of a giant cavity with open bronchus following thoracoplasty combined with extensive apicolysis. Revisions are successful in one-third of the cases. Muscular flap has given 50 per cent negative sputum but it is a procedure still fraught with dangerous complications.

COMMENT

In going over the literature one is impressed by the fact that very little progress has been accomplished in the knowledge of tuberculous cavities during the last ten years. In fact nothing new has been added to the remarkable symposium on this subject held in the Annual Convention of the German Tuberculosis Society in June, 1927 (Bad Salzbrum). To the papers read in that symposium by Baemeister, Hübschmann, May, Felix, and others nothing has been added during the years elapsed since that time.

In every one of these papers one finds the same conclusions which have been formulated a year later by Jaquerod in his excellent atlas on Speleology (study of cavities); it is of interest to quote this author verbatim (p. 13). "A great number of questions," he says, "remain still unanswered, especially concerning the causes and mechanism of spontaneous closure of tuberculous cavities. The puzzle and mystery of these cures will be eliminated only when we have learned better the biological factors which are involved in these cures. For the time being we are forced to be contented with theories which are not satisfactory." We can only say that cure is accomplished by transformation of virulent tubercle bacilli to avirulent, that is, to bacilli which, if they are not dead, are stabilized in a status which renders them unable to grow. Thus clinical cure consists in the transformation of bacilli from "virus" to "vaccine."

We have endeavored to present in our study what we believe to be the answer to these questions and the solution of this puzzling and mysterious problem of clinical cure of tuberculous cavities. We have shown the importance of two fundamental factors, namely, bronchial obstruction with the ensuing atelectasis and the strict aerobism of human tubercle bacillus. We believe that the puzzle and mystery were simply due to the fact that during the long period in which an exclusive and intensive morphologic study of pulmonary tuberculosis was carried out it has been forgotten that both the host, the lung, and the guest, the bacillus, are living organisms, obeying fundamental physiologic and biologic laws. Study of the post-mortem specimen shows only the end result of the fight between the tissues and the bacilli but cannot give us any adequate information upon the physiologic and biologic factors involved in this fight or of the intimate causes of victory or defeat. Likewise the best historian cannot uncover the causes and forces involved in a war by simply studying the battlefield after the war is over.

We further believe that the placing of the study of tuberculous cavities, upon these tangible and solidly grounded foundations, namely, atelectasis of the lung with its mechanical physiologic consequences and the physiology of tubercle bacillus and its biologic corollaries opens new approaches to this fascinating problem. It seems reasonable to believe that more rapid progress in our knowledge on cavities will be accomplished by following strict physiologic and biologic methods of investigation, instead of devising fancy and rather metaphysical explanations. The puzzling and mysterious elements in the behavior of the cavities disappear when we consider them as a problem of pathologic physiology and biology and we attack it with the appropriate methods of research. Likewise the problem of atelectasis was submerged in a haze of contradictory theories and conceptions until it was solved by pure physiologic investigation. Moreover, this investigation had shown us the relation of atelectasis with pulmonary tuberculosis. Ten years ago in concluding our studies in atelectasis we have said[36] that "before long this syndrome will constitute the most important chapter in pulmonary pathology in general and more especially in pulmonary tuberculosis." This prophecy seems to have come true.

Because of the importance of the subject and the new ideas presented in this paper we have deemed it necessary to add a detailed bibliography in order to facilitate the work of the students of this subject.

CONCLUSIONS

A study of giant tuberculous cavities of the lung was presented. The pathology, pathologic physiology, and mechanics of these cavities were studied.

Giant cavities were divided into tension cavities and open cavities. The importance of the patency, stenosis and closure of the draining bronchus was emphasized and discussed at length.

The term "blocked cavities" should be discarded because it does not specify whether the cavity is open or closed.

The findings of direct examination of giant cavities in the living patient by means of cavernoscopic exploration, a new procedure devised for that purpose, were given and discussed.

A new method for surgical closure of cavities which had resisted to thoracoplasty was presented and its results discussed. This procedure consists in the opening of the cavity and filling it with a pedunculated muscular flap.

REFERENCES

1. Alexander, H.: Zum Problem d. Tuberkulöser Kavernen, Beitr. z. Klin. d. Tuberk. **86**: 424, 1935.
2. Alexander, H.: Die Diagnose der Tuberkulöser Kavernen, etc., Fortschr. a. d. Geb. d. Röntgenstrahlen **55**: 1, 1937.
3. Alexander, J.: The Collapse Treatment of Pulmonary Tuberculosis, Baltimore, 1937, Charles C. Thomas, Publisher.

4. Amberson, J. B.: The Nature and Differentiation of Pleural Annular Shadows, Am. J. Roentgenol. **12:** 438, 1924.
5. Amberson, J. B.: The Significance of Roentgenographic Mutations of the Lesions of Chronic Pulmonary Tuberculosis, Am. Rev. Tuberc. **10:** 591, 1922.
6. Ameuille, P.: Etudes des Cavernes Tuberculeuses, de Laënnec a nos jours, Progrès méd. **2:** 1926, 1933.
7. Ameuille, P.: Fermeture spontannée d'une veille cavité tuberculeuse, Bull. et mém. Soc. méd. d. hôp. de Paris **52:** 810, 1936.
8. Ameuille, P.: Injection transpariétale de lipiodol dans une cavité pulmonaire, Bull. et mém. Soc. méd. d. hôp. de Paris **48:** 791, 1924.
9. Ameuille, P., and Levesque: Bronche de drainage des cavernes tuberculeuses, Bull. et mém. Soc. méd. d. hôp. de Paris **47:** 612, 1927.
10. Ameuille, P., Coste, M., and Le Melletier, J.: Cavernes plurilobulaires, Bull. et mém. Soc. méd. d. hôp. de Paris **50:** 449, 1934.
11. Ameuille, P., and Le Melletier, J.: Cavernes géantes en Lames, Bull. et mém. Soc. méd. d. hôp. de Paris **50:** 793, 1934.
12. Ameuille, P., Delhomme, and Raust: La bronche de drainage des cavernes tuberculeuses (étude anatomique), Ann. d'anat. path. **13:** 849, 1936.
13. Ameuille, P., and Shield, J.: Le drainage des cavernes pulmonaires, Studia Tbc. Prag. **1:** 7, 1936.
14. Andral, P.: Cicatrisation des Excavations tuberculeuses, Clinique méd. Vol. 1, Ed. 3, 1826.
15. Bacmeister, A.: Das Kaverneproblem in Seineu Klinischen Bedentung, Beitr. z. Klin. d. Tuberk. **67:** 157, 1927.
16. Baum, F., Mebel, S., and Kane, A.: The Fate of Tuberculous Cavities, Am. Rev. Tuberc. **18:** 596, 1928.
17. Bernard, E., Herrenschmidt, J., and Morin, M.: Cavernes pulmonaires présentant des variations de taille trés accentuées et trés rapides. De la contraction lobaire active, Rev. de la Tbc. **2:** 813, 1936.
18. Bernou, A.: Cavernes a pronostic benin, Gaz. Med. Nantes **15:** 1925.
19. Bernou, A., and Vivant, F.: Des distention cavitaires aigües, Rev. de la tuberc. **5:** 943, 1936.
20. Borgans, H.: Zur Frage der Spontanheilung Tuberkulöser Kavernen, Ztschr. f. Tuberk. **75:** 12, 1936.
21. Borotolotti, R.: Su due casi di guarigionr spontanea di caverne tuberculosi, Lotta contro la tuberc. **3:** 178, 1933.
22. Bronkhorst, W.: Neue Deutungen der Kavernenheilung, Beitr. Klin. d. Tuberk. **72:** 36, 1929.
23. Brooks, W. D. W.: Ancillary Method in the Treatment of Pulmonary Tuberculosis, Brit. J. Tuberc. **32:** 14, 1938.
24. De la Camp, F.: Die prognostische Bedeutung der Kavernen, Beitr. z. Klin. f. Tuberk. **50:** 281, 1922.
25. Kapuszewski, A.: Atelcetase by Lungentuberkulose, Ztschr. f. Tuberk. **75:** 49, 1936.
26. Cardis, F.: L'atélectasie pent-elle resulter de la contraction du poumon? Bull. et. mém. Soc. méd. d. hôp. de Paris **52:** 259, 1936.
27. Castello, V.: Sulla guarigione spontanea delle Caverne tubereolari, Riv. di pat. d. app. respir. **3:** 241, 1934.
28. Castillo, J.: Direct Transthoracic Action on Pulmonary Cavities, Arch. de med. inf., Havana **1:** 463, 1935.
29. Coryllos, P. N.: Importance of Atelectasis in Pulmonary Tuberculosis, Am. Rev. Tuberc. **28:** 1, 1933.
30. Coryllos, P. N.: How Do Rest and Collapse Treatment Cure Pulmonary Tuberculosis, J. A. M. A. **100:** 480, 1936.
31. Coryllos, P. N.: Pathologic Physiology of the Tuberculous Lung. In Goldberg's Clinical Tuberculosis, Vol. 1, p. 151, Philadelphia, F. A. Davis Company.
32. Coryllos, P. N.: Mechanics and Biology of Tuberculous Cavities, Am. Rev. Tuberc. **33:** 639, 1936.
33. Coryllos, P. N.: Pathologic Physiology and Mechanics of Selective Collapse of the Tuberculous Lung (Critical Study), Quart. Bull. Sea View Hosp. **2:** 3, 1937.
34. Coryllos, P. N.: A New Conception of the Mechanics of Cough (Action of the Diaphragm in Cough), Med. Clin. of North Amer., Nov., 1936.

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35. Coryllos, P. N.: Surgical Treatment of Uncollapsed Giant Tuberculous Cavities by Implantation of Pedunculated Muscular Flap, Quart. Bull. Sea View Hosp. **3**: 149, 1938.
36. Coryllos, P. N., and Birnbaum, G. L.: Studies in Pulmonary Gas Absorption in Bronchial Obstruction, Am. J. M. Sc. **183**: 317, 347, 1932.
37. Coryllos, P. N.: Alveolar Gas Exchanges and Atelectasis, Arch. Surg. **21**: 1214, 1930.
38. Coryllos, P. N., Konterwitz, H., and Levine, E.: The Clinical Application of Gas Analysis in Artificial Pneumothorax, Am. Rev. Tuberc. **26**: 153, 1932.
39. Courcoux, A., and Gilson, A.: L'Effacement des immages radiologiques du poumon, Rev. de la tuberc. **8**: 379, 1927.
40. Courcoux, A., and Lermoyer: Caverne Tuberculeuse Occupant Touts L'etendue d'un Poumon, Ann. de Path., October, 1919.
41. Dahlstedt, H.: Beitrage zur Kentniss der localisierte Pneumothorax, Beitr. z. Klin. d. Tuberc. **52**: 79, 1922.
42. Delore, G.: L'etat actual de nos idées sur l'evolution des cavernes tuberculeuses, Progrès méd. 314, 1935.
43. Derscheid, G., and Toussaint, P.: La fermeture des cavernes tuberculeuses aprés phrenicectimic. Rechutes Mechaniques et biologiques, Rev. de phtisiol. **15**: 419, 1934.
44. Derscheid, G., and Toussaint, P.: Guérison de Cavernes tuberculeuses, Rev. Belge de la tuberc. **28**: 175, 1937.
45. D'Hour, H., Delcour, J., and Langeron, L.: Cavernes tuberculeuses totale du poumon gauche, Lyon méd. **140**: 402, 1927.
46. Eloesser, L.: Blocked Cavities in Pulmonary Tuberculosis, J. THORACIC SURG. **7**: 1, 1937.
47. Erofeev, P.: Changes of the Form of the Bronchi and Relation of Bronchi With the Cavities in Pulmonary Tuberculosis (Russian), Arch. Path. & Physiol. **1**: 84, 1935.
48. Faragó, F.: The Presence of *B. Welchii* in Tuberculous Cavities, Am. Rev. Tuberc. **32**: 452, 1935.
49. Faragó, F.: The Spore-Bearing Anaerobic Bacterial Flora of the Cavities, Am. Rev. Tuberc. **32**: 446, 1935.
50. Fales, L. H., and Beudet, E. A.: The Healing of Tuberculous Cavities, Am. Rev. Tuberc. **23**: 690, 1931.
51. Felix, W.: Chirurgische Behandlung der Kaverne, Beitr. z. Klin. d. Tuberk. **67**: 199, 1927.
52. Fleischner, F.: Atelectasen und Lungentuberculose, Beitr. z. Klin. d. Tuberk. **85**: 313, 1934.
53. Gaillard, C.: Un cas de guérison de Caverne, J. de méd. d. Lyon **9**: 320, 1928.
54. Galland, M.: Scléroses pulmonaire, Lausanne, 1920, Thesis.
55. Garcia, y Garcia-Minon, and Julian, Luis: Zur Studien der Tuberculöser Riesenkavernen, Beitr. z. Klin. d. Tuberk. **87**: 509, 1935.
56. Giegler: Ueber die Vorgänge der Reinigung und Heilung der Kavernen und deren prognostischen Bedeutung, Beitr. z. Klin. d. Tuberk. **60**: 195, 1935.
57. Gilbert, M.: Deux Observations Anatomo-cliniques concernant la guérison des cavités tuberculeuses, Rev. de la tuberc. **3**: 979, 1931.
58. Girbal, M.: Traitement des cavernes tuberculeuses géantes, Progrès méd. p. 609, April 13, 1935.
59. González Warcalde, J.: Beitrag zur Histologie Acuter Kavernenbildung, Beitr. z. Klin. d. Tuberk. **89**: 273, 1937.
60. Gräff, S.: Ueber die Bedeutung der Enteilung der Lungenphtisie nach pathologisch-anatomshe gesiehtspunkte, Ztschr. f. Tuberc. **34**: 683, 1921.
61. Hart: Ueber die Heilbarfeit und Heilung Tuberkulose Lungen-Kavernen, Ztschr. f. Tuberk. **35**: 253, 1921.
62. Hautefeuille, M. E.: A propos du traitement des Volumineuses excavations du lobe supérieur, Rev. de la tuberc. **2**: 1090, 1934.
63. Hautefeuille, M. E.: Influence de la situation d'une lision pulmonaire sur les possibilités de la guérison, Rev. de la tuberc. **5**: 298, 1936.
64. Holcomb, F. W., and Weber, C. W.: Atelectasis and the Disappearance of Cavities, Am. Rev. Tuberc. **30**: 299, 1934.
65. Hübschmann, P.: Ueber Kavernen und Pseudo-Kavernen, Beitr. z. Klin. d. Tuberk. **67**: 186, 1927.

66. Hudson, B.: Spontaneous Healing of Cavities. Influence of Altitude, Brit. J. Tuberc. **31**: 18, 1937.
67. Hudson, B.: Interesting Case of Spontaneous Closure of Giant Cavity, St. Barth. Hosp. J. **44**: 108, 1937.
68. Huriau, P. H.: Les cavernes pulmonaires géantes, Paris, 1935, Thesis.
69. Izzo, R. A., Aguillar, O., and Shiffrin, D.: Extra-Giant Cavity Developing Within a Few Days, Semana med. **2**: 1821, 1934.
70. Jacobaeus, H. C.: The Significance of Lung Atelectasis in Tuberculosis, Acta tuberc. Scandinav. **10**: 1, 1936.
71. Jaccound: Curabilité de la Phtisie Pulmonaire, Paris, 1881, Masson & Cie.
72. Jacobs, M.: The Use of Metaphen in Pulmonary Cavities, Am. Rev. Tuberc. **25**: 342, 1932.
73. Jacobs, M., and Beloff, H. M.: Transthoracic Treatment of Tuberculous Cavities, Am. Rev. Tuberc. **36**: 268, 1937.
74. Jaffe, R. H.: The Pathology of Pulmonary Tuberculosis. In Textbook of Clinical Tuberculosis, Goldberg: Philadelphia, 1935, F. A. Davis Company.
75. Jaquerod, M.: Spéléologie Pulmonaire (Atlas), Paris, 1928, Masson & Cie.
76. Jaquerod, M.: La Caverne "Soufflée." Pathogénie et mécanisme de l'apparition et de la disparition de certaines cavernes, Rev. de la tuberc. **5**: 1102, 1936.
77. Joly, H.: La collapsothérapie hypotensive, Paris, Gaston Doin & Cie.
78. Van Joost, C. R.: Merkwurdige Kaverneheilung, Beitrage zur Kentniss der Frage der Kaverneheilungen, Geneesk. tijdschr. v. Nederl.-Indië **75**: 1865, 1935.
79. Katz, S.: La regression spontannes des cavernes pulmonaires tuberculeuses, Rev. de la tuberc. **4**: 589, 1934.
80. Klauschenz, W.: Ausheilungsmöglichkeit und Heilungvorgange bei der tuberkulöse Fruhkaverne, Köln, 1936, Dissertation.
81. Korol, E.: The Alternatively Appearing and Contracting Tuberculous Cavity, Am. Rev. Tuberc. **31**: 457, 1935.
82. Krampf, F.: Pathologisch-anatomische, klinische und experimentelle Untersuchung ueber Lungenschrumplung, Ztschr. f. Tuberk. **51**: 35, 1928.
83. Kuciuktan, I., and Malossini, S.: Freguenza, morfologia et topographia delle caverne pulmonari in una statistica ospitaliero—Sanatoriale, Riv. di pat. d. app. respir. **3**: 667, 1934.
84. Laennec: Traite d'auscultation médicale, Paris, 1879.
85. Lebsche, M.: Beseitigen Ubergrosser tuberkuloser Lungenholen, Deutsche Ztschr. f. Chir. **243**: 530, 1934.
86. Le Seve, D.: La Guerison Naturelle des Cavernes Pulmonaires Tuberculeuses, Paris, 1926, Thesis.
87. Letulle, M.: La Tuberculose Pleuro-Pulmonaire, Paris, 1916, Norbert Maloine.
88. Loebel, R. O., Shorr, E., and Richardson, H. B.: The Influence of Adverse Conditions Upon the Respiratory Metabolism of Human Tubercle Bacilli, J. Bact. **26**: 139, 1933.
89. Lopez, Bonilla, J.: The Atelectatic Closure of Cavities in Artificial Pneumothorax, Arch. argent. de enferm. d. ap. respir. **5**: 67, 1937.
90. Luzzatto, F. G.: Sulle Immagini Pseudo-Cavernose e Sulla Possibilita di rapida elisione di caverne pulmonari dopo interveni collasotherapici, Radiol. med. **23**: 116, 1936.
91. MacDowell, A.: Injection Treatment of Cavities in Pulmonary Tuberculosis, J. A. M. A. **97**: 1090, 1931.
92. MacDowell, A.: The Treatment of Pulmonary Tuberculosis by Gold and Copper Salts, Am. Rev. Tuberc. **25**: 252, 1932.
93. Mantoux, C.: Evolutions Uniquement Radiologique d'une Cavite Pulmonaire. Caverne-Accordion, Paris méd. **79**: 30, 1931.
94. Martinelli, M.: Le Caverne Pulmonari Isolate Extraapical, Riv. di pat. d. app. respir. **2**: 405, 1933; **3**: 193, 265, 1934.
95. Miller, W. S.: Emphysematous Blebs, Am. J. Roentgenol. **18**: 42, 1927; May, 1926; July, 1927.
96. Minet, J., and Patoir, A.: Recidive in situ d'une caverne tuberculeuse, Echo méd. du Nord. **7**: 579, 1937.
97. Morin, J., and Waitz, R.: Variation de taille rapides et accentuées d'une caverne pulmonaire, Rev. de la tuberc. **3**: 240, 1929.
98. Morin, J.: Effacement des cavernes, Bull. Méd. de Leysin. Jan., 1925.

99. Muller, E.: Ueber das Verschwinden von Lungen Kaverne Tuberkuloser, Ztschr. f. Tuberk. **69**: 1933.
100. Naveau, P.: Du rôle de l'atelectasie dans la collapsotherapie des cavernes tuberculeuses, Bull. et mém. Soc. de méd. de Paris **52**: 253, 1936.
101. Naveau, P., and Pesque, M.: Aspects cliniques et radiologiques de l'atelectasie pulmonaire massive au cours de la tuberculose, Arch. méd.-chir. de l'app. respir. **9**: 239, 1934.
102. Niessen, R.: Die Chirurgische Behandlung der oberlappen Kavernen, Schweiz. med. Wchnschr. **1**: 334, 1937.
103. Novy, F. G., and Soule, M. H.: Respiration of the Tubercle Bacillus, J. Infect. Dis. **36**: 168, 1925.
104. Orth: Einige Bemerkungen zur Frage des Heilbarkeit tuberkulöser Lungenkavernen, Ztschr. f. Tuberk. **35**: 251, 1921.
105. Omodei-Zorini, A.: Caverne pulmonare statiche et caverne dynamiche. Criterii di Valutazione, Riv. de pat. e clin. d. tuberc. **10**: 613, 1936.
106. Ornstein, G., and Ulmar, D.: Classification of Pulmonary Tuberculosis. In Goldberg's Clinical Tuberculosis, Vol. 1, p. 59, Philadelphia, 1935, F. A. Davis Company.
107. Pagel, W., and Robinson, H. J.: Spontaneous Healing of Cavities, Papworth Res. Bull. **1**: 37, 1936.
108. Palacio, J., and Mazzei, E. S.: La Atelectasis Pulmonar, Buenos Aires, 1937, Aniceto Lopez.
109. Pinner, M.: The Cavity in Pulmonary Tuberculosis, Am. J. Roentgenol. **20**: 518, 1928.
110. Pinner, M.: In J. Alexander's Collapse Treatment of Pulmonary Tuberculosis, Baltimore, 1937, Charles C. Thomas, Publisher.
111. Piquet, C. A., and Giraud, A.: La disparition radiologique des Cavernes tuberculeuses, Ann. de méd. **14**: 362, 1923.
112. Piquet, C. A., and Giraud, A.: Paradores du pneumothorax therapeutique, Presse méd. 1924.
113. Pottenger, F. M.: Cavitations in Pulmonary Tuberculosis, Am. Rev. Tuberc. **26**: 215, 1932.
114. Pottenger, F. M.: Interfering Mechanical Factors in the Healing of Pulmonary Tuberculosis, Am. Rev. Tuberc. **26**: 229, 1932.
115. Pruvost, P., Meyer, A., and Livieratos: Du traitement des cavernes géantes par le pneumothorax, Presse méd. **1**: 385, 1935; Arch. méd.-chir. de l'app. respir. **10**: 9, 1935.
116. Rassfeld, L.: Bacteriologische Leichenblutuntersuchungen. Obligaten Anaerobia, Ztschr. f. Hyg. u. infectionskr. **93**: 393, 1921.
117. Reinders: Ueber Form und Grösse der Fruhkacernen, Ztschr. f. Tuberk. **51**: 438, 1928.
118. Rist, E.: La Tuberculose, Paris, 1927, Librarie Armand Colin.
119. Rist, E., and Strohl: La résorption et l'équilibre gaz dans le pneumothorax fermé, Ann. de méd. 233, 1920.
120. Roubier, C.: Caverne tuberculeuse occupant toute l'étendue d'un poumon, Lyon méd. **134**: 332, 1924.
121. Roubier, C., and Doubrow, S.: Etude histopathologique du mode d'action du pneumothorax artificiel dans la Tuberculose pulmonaire, Rev. de la tuberc. **10**: 463, 1929.
122. Salkin, D., Cadden, A. V., and McIndoe, R. B.: The Blocked Pulmonary Cavities, Am. Rev. Tuberc. **34**: 634, 1936.
123. Salkin, D.: Post-Mortem Bronchography, Am. Rev. Tuberc. **34**: 649, 1936.
124. Sampson, H. L., Heise, F. H., and Brown, L.: A Study of Pulmonary and Pleural Annular Shadows, Am. Rev. Tuberc. **2**: 664, 1919.
125. Sayé, L.: Examen radiologique des cavernes, Arch. Espan. de Phtisie, Jan., 1919-May, 1920.
126. Sayé, L.: Sur les formes cliniques d'atelectasie pulmonaire, Bull. et mém. Soc. méd. d. hôp. de Paris **3**: 52, 272, 1936.
127. Schminke, A.: Lungenkavernen, Beitr. z. Klin. d. tuberk. **67**: 124, 1927.
128. Scorpati, G., and Berthet, E.: Sur la fausse interpretation de certains aspects radiologique, Presse méd. **1**: 565, 1935.
129. Scorpati, G.: Pseudocaverne pulmonari da emphysema bolboso, Rassegna internaz. di clin. e terap. **34**: 252, 1935.

130. Sechi, G., and Bassoni, V.: Le caverne tubercolare del polmone; contributo allo studio de la scompars a spontanea di caverne stud, Clin. e Soc. Tisiol. **4**: 136, 1935.
131. Sergent, E., and Bordet, F.: Diagnostie differentiel des pneumothorax enkystés et des grandes cavernes, Bull. et mém. Soc. méd. d. hôp. de Paris **48**: 1076, 1924.
132. Sergent, E., and Moizesco: Nettoyages Radiologiques, Rev. de la tuberc. **8**: 401, 1927.
133. Shipman, J.: Bronchial Factor in Cavitations, Am. Rev. Tuberc. **38**: 337, 1938.
134. Sisti, M. A.: Sulla Valutazione di alcuni fattori Mecanici della cavernogenesi, Riv. di pat. e clin. d. tuberc. **10**: 620, 1936.
135. Stephani, J.: Characteres radiographiques cavities qui cicatrisent spontanuément, Arch. d'électric méd. **45**: 155, 1937.
136. Stephani, J., and Delore, P.: Les cavernes pulmonaires tuberculeuses, Paris, 1927, Gaston Doin & Cie.
137. Strandgaard, N. J.: Spontaneous Healing of Cavities, Hospitalstid. **78**: 533, 1935.
138. Tchertkoff, I. G., and Bobrowitz, I. D.: Spontaneous Closure of Tuberculous Pulmonary Cavities, Quart. Bull. Sea View Hosp. **1**: 173, 1936.
139. Tapia, M., Jorda, R., and Tapia, J.: El Factor atelectatieo on la tuberculosis pulmonare, An. de med. int. **3**: 397, 1934.
140. Toniolo, G.: Sulle immagini pseudo-cavernose, Radiol. med. **22**: 1013, 1935.
141. Trocmé, C.: Etude historique et statistique sur les preuves anatomo-pathologiques de la guérison de la tuberculose pleuro-pulmonaire, Paris, 1903, Thesis.
142. Trocmé, C.: Essai sur les scléroses pulmonaire, Ann. de méd. **40**: 106, 1936.
143. Turban and Straub: Kavernendiagnose und Kavernenheilung, Ztschr. f. Tuberk. **48**: 81, 1925.
144. Wolf, J. E.: Der ableitung-bronchus Tuberkulöser kavernen, Beitr. z. Klin. f. Tuberk. **66**: 700, 1927.
145. Zainál: Spontaneous Cures of Tuberculous Cavities, Geneesk. tijdschr. v. Nederl.-Indië **1**: 146, 1937.
146. Zeissler, J.: Handbuch d. Pathol. Microorg. 4: 1099, 1928.

EXTRAPLEURAL THORACOPLASTY

Further Experiences With a Muscle Splitting Operation

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Chicago, Ill.

OVER the past decade the operation of extrapleural thoracoplasty has been gradually extended and improved. To obtain a high percentage of permanent results it has been found necessary to remove longer and longer segments of ribs, to resect the transverse processes, and finally to perform simultaneously an extrafascial apicolysis. Only when all of these things are done is a collapse obtained which approaches Brauer's requirement—that it equal that afforded by a good pneumothorax. While the new operation has increased the cures from 40 to 80 per cent of those operated upon, it has also become much more difficult to perform and much more trying both to the surgeon and the patient. Shock and paradoxical respiration are more common complications, deformity is greater, and in some clinics it has been found that the exposure of such broad surfaces of tissue far from one and one-half to two hours has increased the incidence of wound infections.

To avoid these drawbacks I have recently modified and readopted a muscle splitting operation which I first described in 1928.* In this the full length of the upper six ribs and long segments of the lower ones are removed through multiple small muscle splitting incisions, much of the stripping of the ribs being done subcutaneously with specially devised periosteotomes (see Fig. 1). An extrafascial apicolysis is performed at the time of the removal of the posterior segments of the upper three ribs. The great advantage of the operation is the marked reduction in postoperative reaction. The pulse rarely rises above 100 and it is not uncommon for the patients to sit up for the evening meal the day of the operation and even walk to the toilet.

By destroying the periosteum of the posterior portions of the upper six ribs, regeneration does not rush the completion of the operation, and the stages may be spaced as far apart as seems advisable. Postoperative pain is minimal and the ultimate function of the arm and shoulder is better than when the muscles have been transected. The operation is particularly useful in patients who, because of advanced

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*Head, Jerome R.: Extrapleural Thoracoplasty: A Muscle-Splitting Operation, Arch. Surg. 16: 1075, 1928.

years or cachexia, are poor surgical risks and for whom the more extensive procedure is particularly dangerous.

STAGE I

The incision for the first stage extends from the junction of the inner and middle thirds of the spine of the scapula upward and backward in the line of the fibers of the trapezius to the first dorsal spine (Fig. 2). The fibers of the trapezius are split and the muscle freed on its under surface to expose the rhomboideus major. This muscle, which here runs downward from the spine at a relatively acute angle, is also split. The loose areolar tissue beneath it is separated by the finger, which can be run well up over the apex of the thorax and well down under the scapula. The serratus posterior superior is cut away and the erector spinae muscles are dissected back toward the spine. Through this exposure 12 cm. of the third rib, most of the second rib,

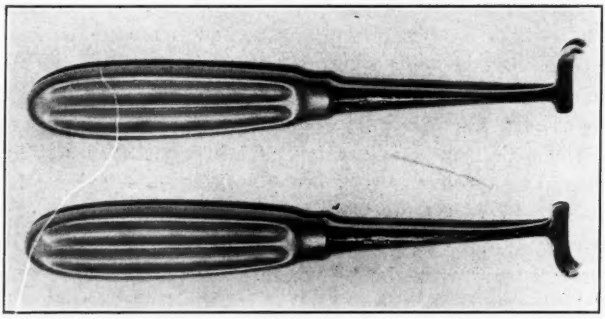

Fig. 1.—Periosteotomes for stripping upper and lower border of ribs.

and the whole of the first can be resected. An extrafascial apicolysis can then be performed in the usual manner and if it seems advisable can be extended at the second posterior operation.

When the retractors are removed, the muscles fall back into place so that their edges can be approximated without tension by a few interrupted silk sutures.

This stage of the operation is the most difficult of all. The exposure is meager and considerable patience is needed to carry it through and secure the necessary collapse.

STAGE II

The second stage can be performed a few days later. A small incision is made below the clavicle in the direction of and just over the second rib (Fig. 3). The fibers of the pectoralis major are separated, and by adjusting the retraction the anterior segments of the second and third ribs and any remaining portion of the first can be reached and removed.

STAGE III

The full lengths of the fourth, fifth, and sixth ribs can be removed at a single stage through two small incisions or the posterior segments

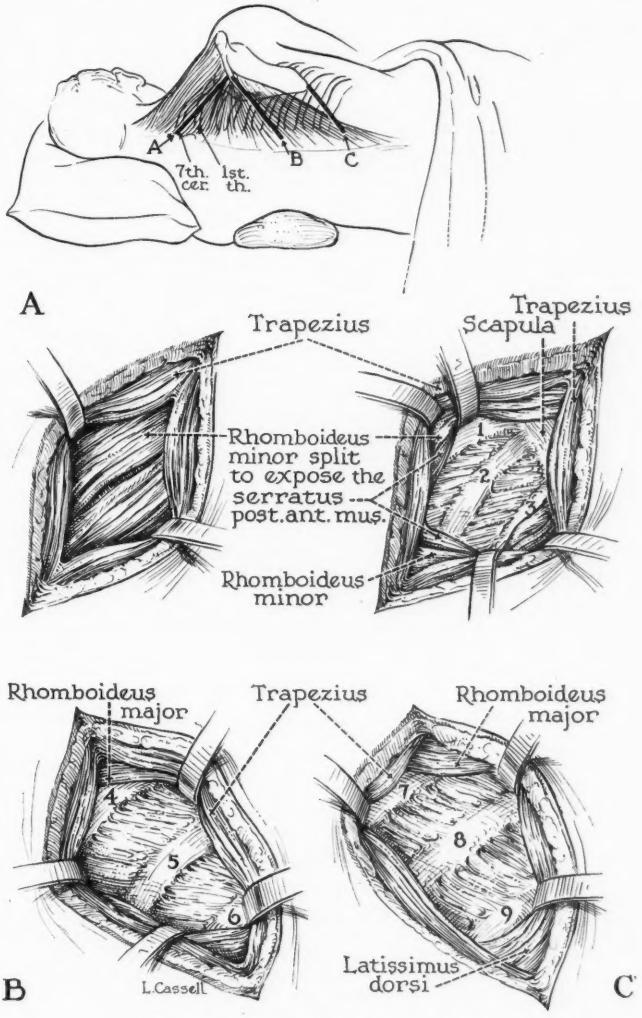

Fig. 2.—Drawings illustrating operation.

at one operation, and the anterior segments, later. The posterior incision extends from the junction of the inner and middle thirds of the spine of the scapula downward and backward almost parallel to this crest to the spinous process of the seventh dorsal vertebra (Fig.

2). The trapezius and rhomboid are split in the directions of their fibers and the posterior segments of the ribs stripped and cut proximally. If the full lengths of the ribs are to be removed at one sitting, a second small vertical incision is made over the fifth rib in the anterior axillary line. The tissues of the chest wall can here be retracted to expose the fourth to sixth ribs, which are stripped forward to the costal cartilage and backward to the point where they have been freed through the posterior incision. Most of the stripping is done subcutaneously. Section of the ribs anteriorly permits them to be drawn out through either the posterior or the anterior opening.

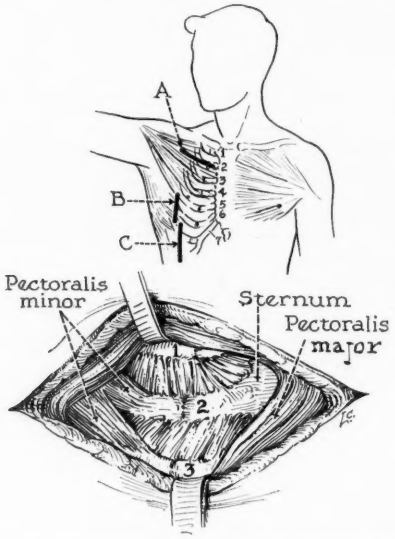

Fig. 3.—Anterior incisions, with detail of the infraclavicular incision.

STAGE IV

Transection of the latissimus dorsi muscle through the conventional hockey stick incision is less traumatizing than mobilization of the scapula for the upper stages and consequently may be used for completion of the thoracoplasty. The same result can be obtained with less trauma and deformity by employing a small muscle splitting incision posteriorly and a second small vertical incision in the anterior axillary line.

COMMENT

Temperature, pulse, and respiration curves showed minimal postoperative reactions.

The type of collapse which can be obtained is shown in Fig. 4.

At the present time it is my practice to use this operation only on the older patients, on those who are poor operative risks, and on those who have no large cavities. The exposure does not permit freeing of the chest wall from the attachments of the serratus magnus and scalenes, and for this reason the collapse is occasionally less complete than where these procedures can be carried out. At present, steps are being taken to overcome this defect in the operation.

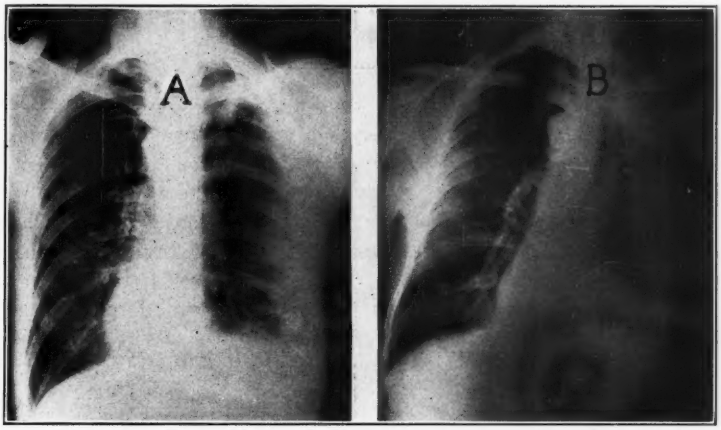

Fig. 4.—*A*, Tuberculosis of the right lung, with a medium-sized cavity at the apex; arrested disease in the left lung, before thoracoplasty. *B*, After completion of thoracoplasty.

SUMMARY AND CONCLUSIONS

1. A modification of the operation of extrapleural thoracoplasty is described in which the required lengths of ribs are removed through multiple small muscle splitting incisions.

2. At the first stage an extrafascial apicolysis can be performed.

3. The operation has the following advantages: (1) The postoperative reaction is minimal; (2) the collapse is produced gradually; (3) the muscles of the shoulder girdle are not transected; (4) it can be used on older persons and those who are in poor condition with greater safety than can the conventional operation; and (5) the incidence of wound infections is less than following the conventional operation.

THORACOPLASTY FOR TUBERCULOSIS AND CHRONIC EMPYEMA THROUGH SHORT INCISIONS

Experiences With a New Method of Rib Removal, Including Description of Anterior Extrafascial Apicolysis*

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THE operation of extrapleural thoracoplasty for tuberculosis has become so standardized an operative procedure that contemplated variations in the technique of its performance demand explanation. He who observes the operation as well as he who performs it will admit readily, however, that it is a radical therapeutic measure which neither patient nor surgeon can regard lightly. Any simplification of the operation which lessens the hazard or extends the indication for performance of the operation to patients ill-suited to withstand the magnitude of the typical procedure needs no further justification. This presentation concerns this phase of the technique of thoracoplasty.

In a previous communication[5] the writer has described how the operation of extrapleural thoracoplasty may be performed through short incisions, the rib being first divided anteriorly and freed along its lower border; at a second posterior operation the rib is again divided, separated from its periosteal bed on its superior border, and removed. It was then suggested that the elaboration of special instruments to facilitate rib removal would probably further simplify the operation. A more extended study of the technique of rib removal has proved such to be the case.

THE NEW TECHNIQUE

The reader is referred to the earlier paper for the plan of operation previously suggested. The principles of the procedure, however, are illustrated well in the accompanying sketches (Fig. 1). The O'Brien periosteal raspatory depicted there was used initially. It is an excellent instrument but when pushed through a long narrow tunnel to which direct vision does not extend, it has obvious limitations which its user is soon to learn. This weakness of the instrument (for which purpose it was not intended by its innovator) lies in the fact that it is a blunt raspatory. In consequence, when unusual resistance is encountered in the separation of the rib from its bed, the instrument

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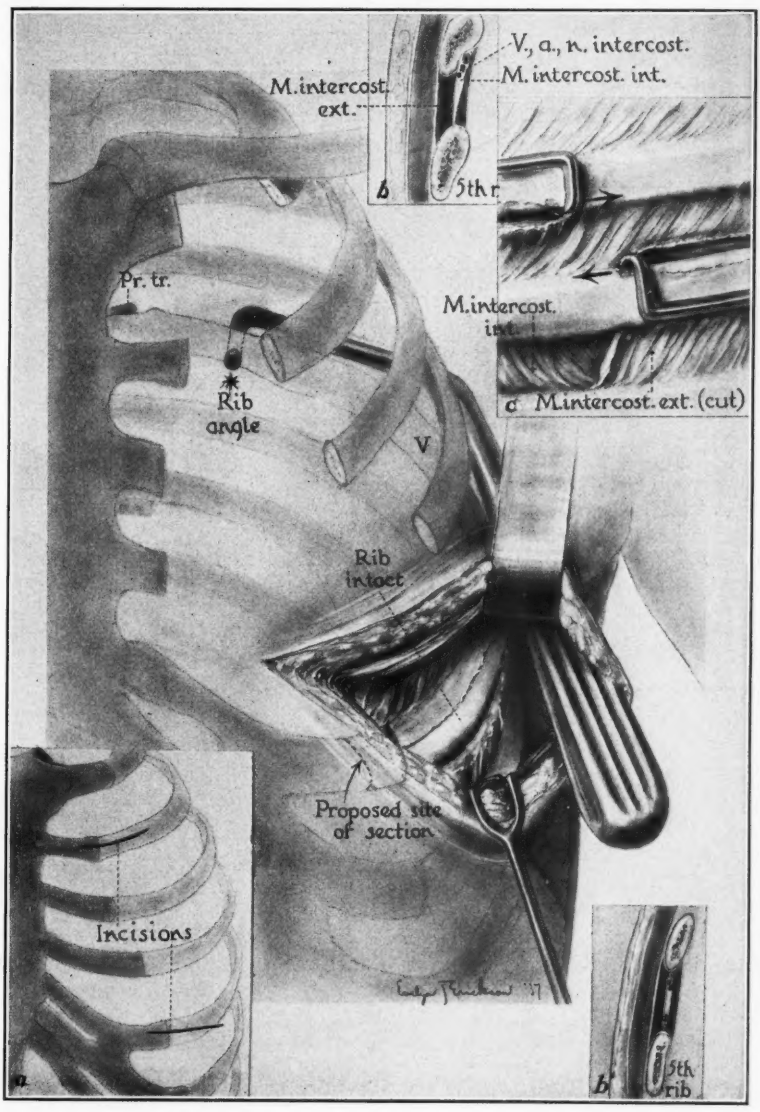

Fig. 1A.

Figs. 1A and B.—The operation as first proposed. Advantage is taken of the direction of insertion of the fibers of the intercostal muscle bundles in the removal of a rib. (Surgery 2: 852, 1937.)

fails to shear off the periosteum but either tears the adjacent muscle attachment, or if considerable force is used, "jumps the track" and leaves the periosteal groove burying the hook of the instrument in the muscles of the chest wall, from which site of lodgment it may be extricated with some difficulty. It was obvious that a more trusty instrument had to be devised to circumvent this difficulty and to insure easy and safe removal of long segments of rib by this method.

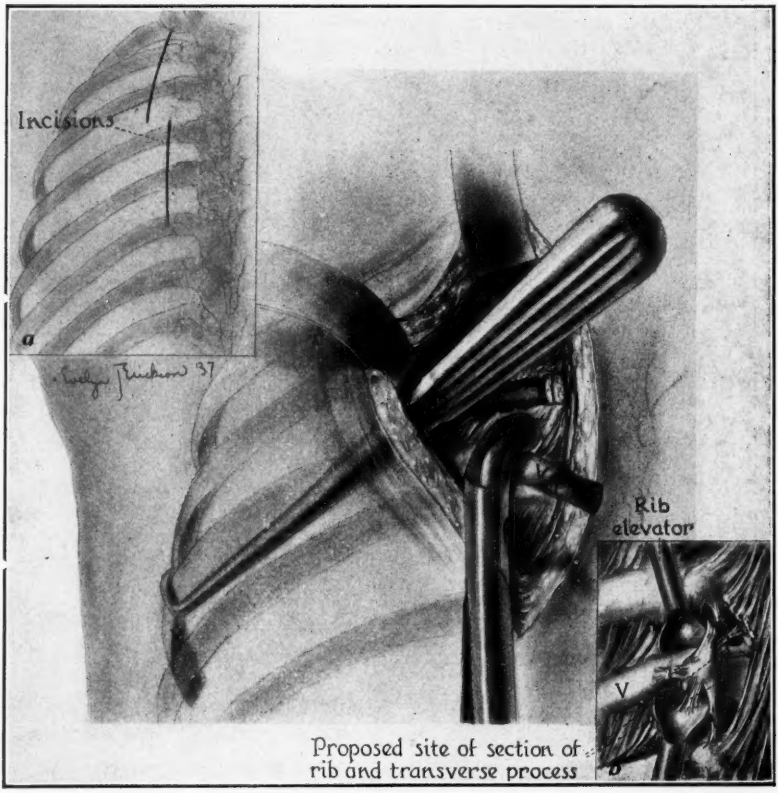

Fig. 1*B*.

The services of Mr. John Phelan of the Scientific Apparatus Shop of the University of Minnesota were enlisted. A number of models of periosteal raspatories were made up as shown in Fig. 2. After many trials in the post-mortem room as well as at the operating table, it became apparent that the secret of uniform success in removing a rib by this method lay in the construction of a sharp instrument without a hook which could be plied subperiosteally. At this juncture all available instrument catalogues were scrutinized carefully for this

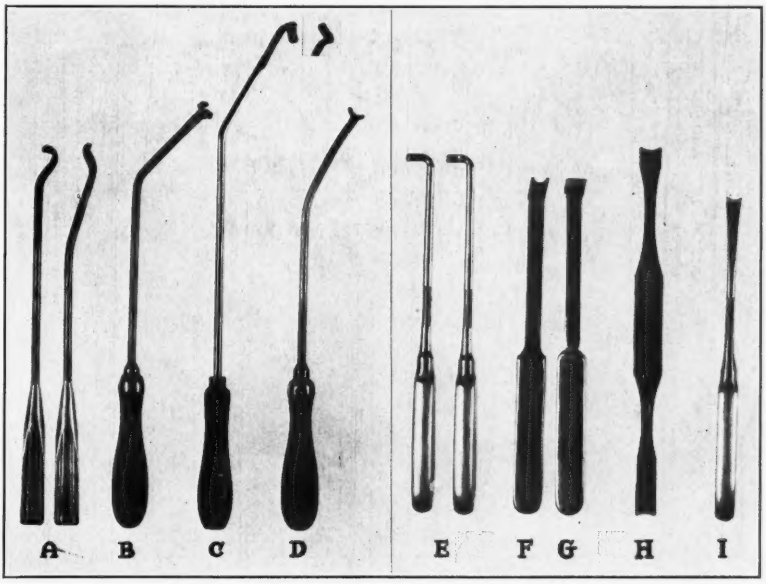

Fig. 2.—*A*, *B*, *C*, and *D* are early models of periosteal raspatories made for me by Mr. John Phelan. These instruments conformed in a general way to hook patterns of periosteal separators. *C* has a swivel hook, held in a stationary position against the shank of the instrument as the raspatory is pushed forward; when it is pulled back, the swivel permits the hook to straighten out and return in the axis of the instrument (suggestion of my associate, Dr. John Paine). This instrument proved fairly satisfactory for the anterior operation when done after the manner shown in Fig. 1. None of these proved very useful. The adoption of sharp periosteal elevators as shown in *E*, *F*, *G*, and *H* has removed the ordinary restrictions relating to the direction in which the rib was to be freed from its bed. The right handed instrument *E* for the top surface becomes the left handed instrument when used on the lower surface, and vice versa. *F* is useful on the posterior segment where the rib is triangular in outline. *G* has the widest range of usefulness and is particularly helpful in freeing the periosteum from the upper surface of the rib, whether the separation is done through an anterior or posterior incision. *H* is used to separate the pleura from the rib on its lower surface, as is also *E*. *I* is the Semb periosteal raspatory, extremely useful in freeing the borders of the rib.

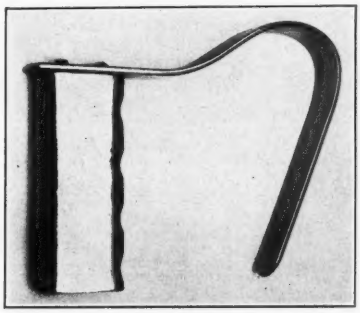

Fig. 3.—Intercostal retractor for elevating structures of the chest wall from the upper surface of the rib while the rib is being freed—useful in both anterior and posterior operations. The raspatory *G* shown in Fig. 2 is shoved over the upper surface of the rib before the blade of this retractor is entered.

type of instrument and a number of unfamiliar patterns were obtained and tried. The notched Semb periosteal raspatory proved eminently practical for freeing the upper and lower borders of the rib, and the periosteal raspatories shown in Fig. 2 served effectually to free the upper and lower surfaces of the rib from its periosteal and muscle attachments. A special retractor with a narrow but long blade was

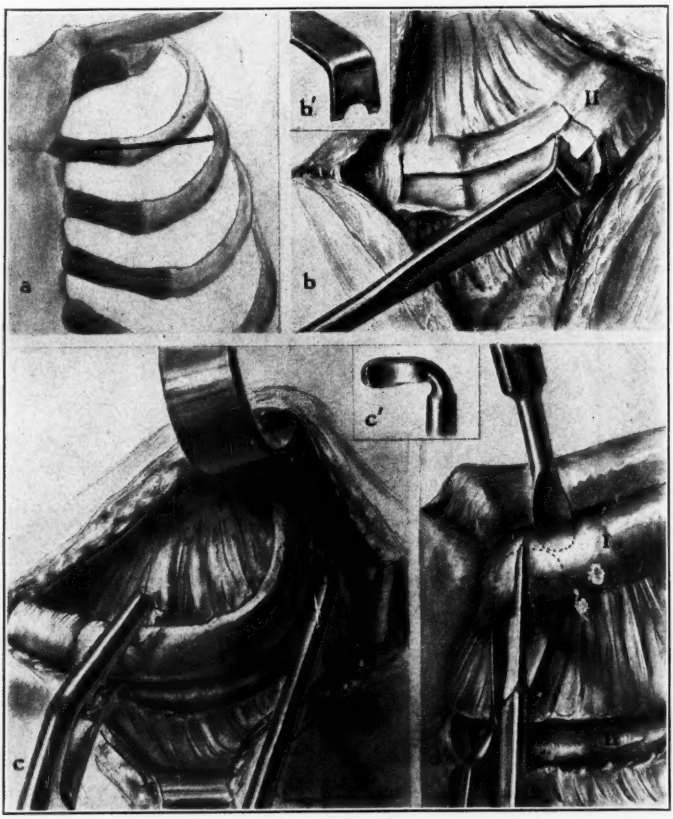

Fig. 4.—The technique of rib removal employing sharp periosteal raspatories. (*a*) The incision for the anterior operation (the skin incision for the posterior operation remains the same as shown in Fig. 1). (*b*) The Semb periosteal raspatory (Fig. 2) is used to free the edges of the rib. (*c*) The cartilage is divided and the raspatories shown in Fig. 2 (*E, F, G,* and *H*) are used to free the upper and lower surfaces of the rib. The intercostal retractor elevates the structures of the chest wall. (*d*) Manner of liberating the first rib; the division of the cartilage is also shown. Occasionally the first rib cartilage is ossified and must be cut with rib shears. In the performance of anterior extrafascial apicolysis (Fig. 8), if the lung is adherent anteriorly its mobilization may be facilitated by rongeuring away more cartilage or even a portion of the sternum.

devised also to elevate the muscles of the chest wall off the rib during separation from its periosteal bed* (Fig. 3).

*The retractor and periosteal raspatories described here are now available through the Pilling Company.

The technique of rib removal with employment of these sharp periosteal separators is shown in Fig. 4.* It became quickly apparent that an additional great advantage of the method was the fact that the rib, when previously divided, could be removed with equal ease from either end. How long a segment may be removed from before or behind is shown in Fig. 5. In brief, the rib when divided anteriorly can be removed in its entire length from behind. When divided posteriorly, however, the short segment containing the sharp curve of the angle must also be excised simultaneously, if the rib is to be removed later through an anterior incision.

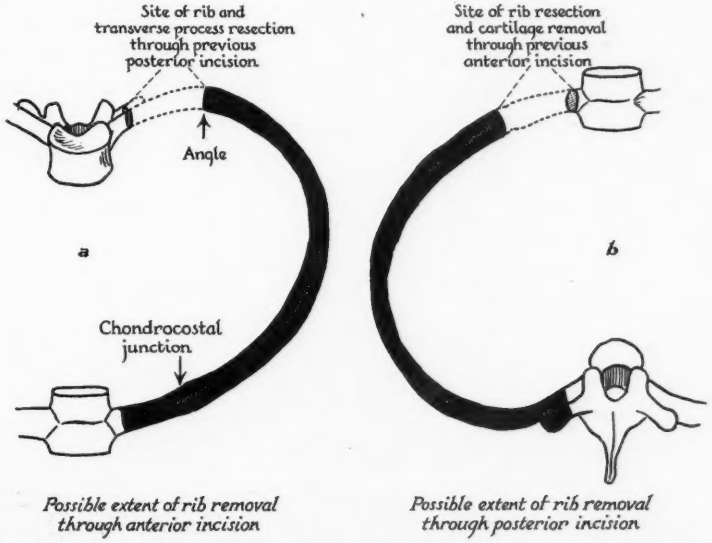
Site of rib and transverse process resection through previous posterior incision
Angle
a
Chondrocostal junction
Possible extent of rib removal through anterior incision
Site of rib resection and cartilage removal through previous anterior incision
b
Possible extent of rib removal through posterior incision

Fig. 5.—The possible extent of rib removal through an anterior or posterior incision when sharp periosteal raspatories are employed.

THE USUAL PLAN OF PROCEDURE

With the patient lying supine, a short incision is made over the second rib anteriorly, and the costal cartilages of the first, second, and third ribs are removed together with quite liberal portions of the ribs themselves, as shown in Fig. 4. The first rib is cut behind the groove for the subclavian artery. For several months the writer has been performing an extrafascial apicolysis (to be described below) at the time of the anterior operation, a procedure demanding wider excision of the anterior segments of rib. About ten to fourteeen days later (a somewhat longer interval of time than first suggested) the residual

*The illustrations made for me by Miss Evelyn Erickson of the Department of Medical Arts of the University of Minnesota.

segments of the upper three ribs together with the transverse processes are removed through a vertical incision from behind. The patient lies prone, his head suspended as indicated in Fig. 6. The scapula is not visualized in the posterior operation performed by this method, and there exists no necessity to retract it. At this operation, the fourth, or the fourth and fifth, or even the sixth and seventh ribs may be divided also, and their short angular paravertebral segments removed. By cutting and removing the transverse process as shown in Fig. 7, the neck of the rib can be freed subperiosteally obviating injury

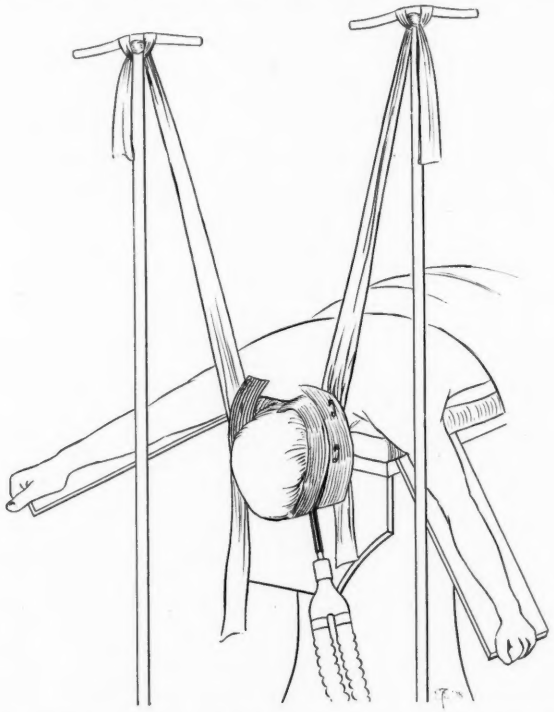

Fig. 6.—A satisfactory manner of suspension of the patient's head for the anterior operation (suggestion of Dr. Ralph T. Knight, University Hospital anesthetist). This simple improvised expedient allows the anesthetist to manipulate the patient's head, which the cerebellar head rest employed previously for the same purpose did not. Intratracheal anesthesia may be given as shown here, or the mask may be used (cyclopropane anesthesia).

to the intercostal vessels. If both transverse process and rib are cut at once, wounding of the intercostal vessels is much more likely.

If only the residual stumps of the upper three ribs are removed at the time of the posterior operation, a relatively long interval may be allowed to intervene, if deemed necessary, before the performance of the next stage of rib resection, which would be performed also through

a short transverse anterior incision followed in turn by posterior rib removal ten to fourteen days later. If, however, additional ribs are cut at the time of the posterior operation, these ribs should then be removed through an anterior incision ten to fourteen days later. Through employment of this scheme of operation, adequate rib removal may frequently be accomplished in three operations, particularly when apicolysis is done. Whereas in the beginning I was anxious to observe the effect of wide excision of anterior segments of rib and removed not infrequently the cartilages of the fourth, fifth, sixth, and even the seventh ribs at the time of the second anterior operation (even on the left side), I do not believe that such excision aids materially in the collapse of apical cavities. In consequence I now excise shorter segments of the fourth to seventh ribs, conforming more to the conventional extent of rib resection for these segments. At the time of the second anterior operation if three ribs are to be excised

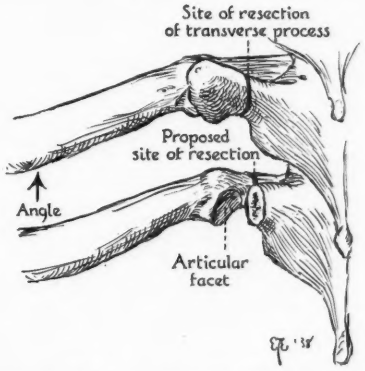

Fig. 7.—Present method of accomplishing section of transverse process and rib posteriorly. Compare with similar insert in Fig. 1*B*. Accidental division of the intercostal vessels may be avoided constantly if the transverse process is divided and removed before the rib is cut. The preliminary removal of the transverse process permits subperiosteal preparation of the rib proximal to the articular facet.

a short somewhat oblique incision tapering posteriorly at its lower end is made in the axillary line between the pectoralis major muscle anteriorly and the latissimus dorsi posteriorly. If four ribs are to be excised two separate horizontal buttonhole incisions offer adequate exposure.

The average interval between the first operation and the last in a small group of patients who have had six or seven ribs removed in their entire lengths in four operations has been twenty-seven days. If at the time of the posterior excision of the residual stumps of the first upper three ribs, additional ribs are sectioned as described above, an adequate collapse can be obtained usually in three stages. The length of time between the first and last operation has remained, however, about the same as in the group in which four operations were

used to achieve the same extent of rib removal. About the first week of last December, I began to employ the sharp periosteal raspatories described herein, and at the same time began to perform extrafascial apicolysis through an anterior incision to be described below. The interval of time between operations, in the meanwhile, has been lengthened somewhat, but I now usually accomplish the same extent of rib removal in three operations for which four were employed previously. In a recent small group of cases who have had apicolysis as well as excision of six or seven ribs (total excision of upper three and subtotal of fourth, fifth, sixth, and seventh) by the method herein described, the average interval between the first and last operation has been thirty-one days.

Fig. 8.—Parasternal bone regeneration from perichondrium of second, third, and fourth costal cartilages (right side) after complete excision of ribs and cartilages.

SUPERIORITY OF THIS METHOD OF RIB REMOVAL

Apart from the avoidance of division of large muscle bundles as is of necessity done in the classical operation of extrapleural thoracoplasty, the chief advantage of the method lies in the staging of rib removal. When the removal of three ribs is divided between an anterior and a posterior operation, approximately one and one-half ribs are removed actually at each sitting. In the prior communication upon this subject the impression may have been lent that division of the muscles in the typical operation of thoracoplasty, employing a long incision, rather than rib removal is responsible for the occurrence of dyspnea. This is not strictly true, for I have now seen a few

instances of dyspnea attend removal of long segments of the lower ribs in patients with a thin pleura by the method described here. The reactions attending this type of operation, in the main, are considerably less than that attending the more formal operation of thoracoplasty. With employment of the sharp rib raspatories, the ribs may almost uniformly be removed without a vestige of muscle fiber remaining on them. In consequence, bleeding, of course, is minimal. With the proper staging of operative procedures and the postoperative employment of intratracheal catheter suction to avoid as well as treat atelectasis as suggested by Haight, this method of rib removal should offer a means of performing extrapleural thoracoplasty with minimal risk and optimal collapse. Elevation of the shoulder, a not uncommon accompaniment of the long posterior incision, apparently does not attend the procedure here described.

As has previously been stated, firm anterior chest walls attend excision of the costal cartilages.[6] In a patient reoperated upon within the past year because of persistence of an apical cavity despite total removal of the upper four ribs and cartilages and excision of lesser lengths of the fifth, sixth, and seventh ribs a year previously, the parasternal bone regenerated from the perichondrium was excised (Fig. 8). No instances of infirm anterior chest walls have been seen in this clinic over a four-year period, during which time the upper three and occasionally the fourth costal cartilages have been removed routinely in the performance of thoracoplasty for tuberculosis.

THE NEED FOR APICOLYSIS

All who have had an experience of any length with thoracoplasty for tuberculosis know that the apex of the lung still retains its attachment above. No matter how complete or excellent the bony collapse of the chest wall, the lung remains suspended at the cupola of the thoracic cage. In many instances, such failure to depress the apex of the lung does not compromise the result. Not uncommonly, however, an upper lobe cavity, though compressed against the spine by the strong lateral pressure of the massive muscles of the chest wall, still remains open. It is rather discouraging to look at x-ray films after thoracoplasty showing an excellent result as judged in the light of diminution of the volume of the thorax, but to note a cavity still persisting in the apical portion of the lung. Surgeons who perform secondary operations on such patients know how difficult the task is. In envisaging the ultimate result of thoracoplasty it is well to bear in mind this somewhat frequent shortcoming of rib removal, continued apical suspension of the lung with potential failure to close the cavity and render the patient noninfective. The group of patients whose physical status has been improved vastly by thoracoplasty but who still remain social derelicts because they are still carriers is probably

not small. A deliberate attempt should be made at the time of the initial procedure, even if at some increased risk, to forestall this occurrence by undoing the apical suspension of the lung. Semb, of Oslo, has shown how this can be done. His own results indicate that the method in his hands does not command any extension of the risk, even though it increases appreciably the magnitude of the operative intervention.

ANTERIOR EXTRAFASCIAL APICOLYSIS

It is not difficult at the time of the anterior chondrocostectomy to perform an extrafascial apicolysis. A personal experience, with performance of twelve posterior extrafascial apicolyses after the method

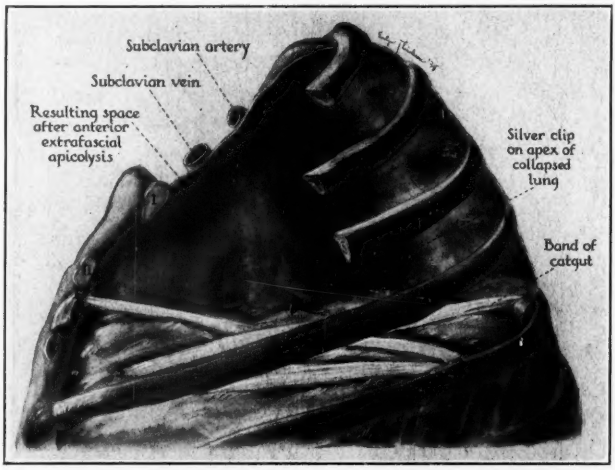
Subclavian artery
Subclavian vein
Resulting space after anterior extrafascial apicolysis
Silver clip on apex of collapsed lung
Band of catgut

Fig. 9.—The technique of extrafascial apicolysis through an anterior incision. The extent of rib removal is also shown.

of Semb and a somewhat lesser number performed through an anterior incision, leads me to believe that the anterior approach presents the advantage of improved vision and ease of performance. The anterior portion of the lung is freed much more readily and effectually through an anterior incision. On encountering a lung fixed rigidly to the adjacent structures in the cupola of the thorax, when performing apicolysis from behind, I have felt very timid about trying to free the lung anteriorly out of range of vision. Moreover, extrafascial apicolysis can be performed ordinarily very satisfactorily through a short anterior incision after removal of the upper three cartilages and portions of the ribs, as indicated in Fig. 9. Even though a lateral film, which should be made routinely, shows the cavity to be largely posterior, my own experience has been that a large cavity may remain open unless

the depression of the anterior portion of the lung has been satisfactory. The extent to which the anterior portion of the lung may be depressed by anterior extrafascial apicolysis (Fig. 9) should help to preclude that occurrence.

TECHNIQUE

The first and second intercostal muscle bundles together with the perichondrium are cut anteriorly near the sternum. Employing the tips of the fingers, or a small sponge mounted on a hemostat, pressure is applied along the lower surface of the second and third ribs, pushing away the periosteum. By this plan of procedure, a plane of cleavage is established laterally and posteriorly outside of the endothoracic fascia. Occasionally the lung is adherent in this plane through thickening of the pleura, fascia, and periosteum, making stripping

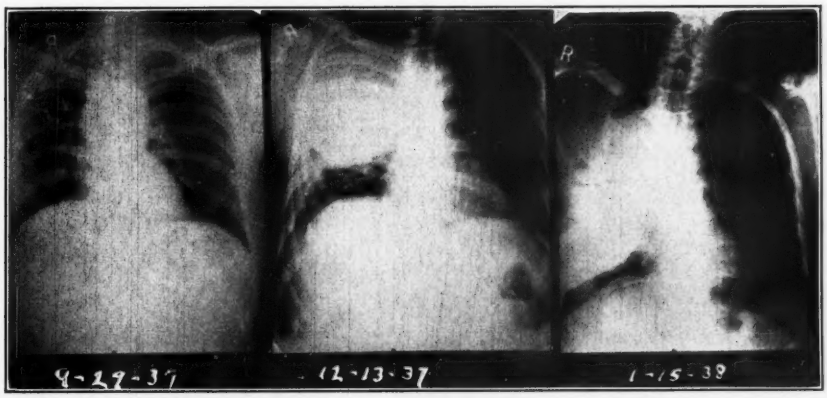

Fig. 10.—Roentgen film before and after anterior extrafascial apicolysis, with complete excision of the upper six ribs and removal of the upper three costal cartilages. The outline of the cavities has been accentuated slightly by tracing the periphery gently with a soft lead pencil. At the time of the operative procedure a silver clip was snapped on the very summit of the lung. The extent of the depression of the lung is apparent in the film on the right.

difficult. Usually, however, this part of the procedure is accomplished with ease because the separation is done along a natural slope of the rib which is easily followed. At this juncture the separation is commenced between the sternum and clavicle and the endothoracic fascia overlying the lung anteriorly. By alternately working from behind and before, the suspension of the lung through the agency of the pleurocostal and pleurovertebral ligaments (Sibson's fascia or suspensory ligaments of Zuckerkandl) can be visualized and these ligaments divided.

On completion of this combined procedure of blunt separation and dissection, when satisfied that the lung has been mobilized adequately or as far as it can conveniently be accomplished, I attempt to hold

it in the depressed position in the manner indicated in Fig. 9. A broad piece of catgut employed by urologic surgeons to suspend a ptosed kidney is woven back and forth over the dome of the lung. Anteriorly, it is fixed to the mesial portion of the perichondrium in the second

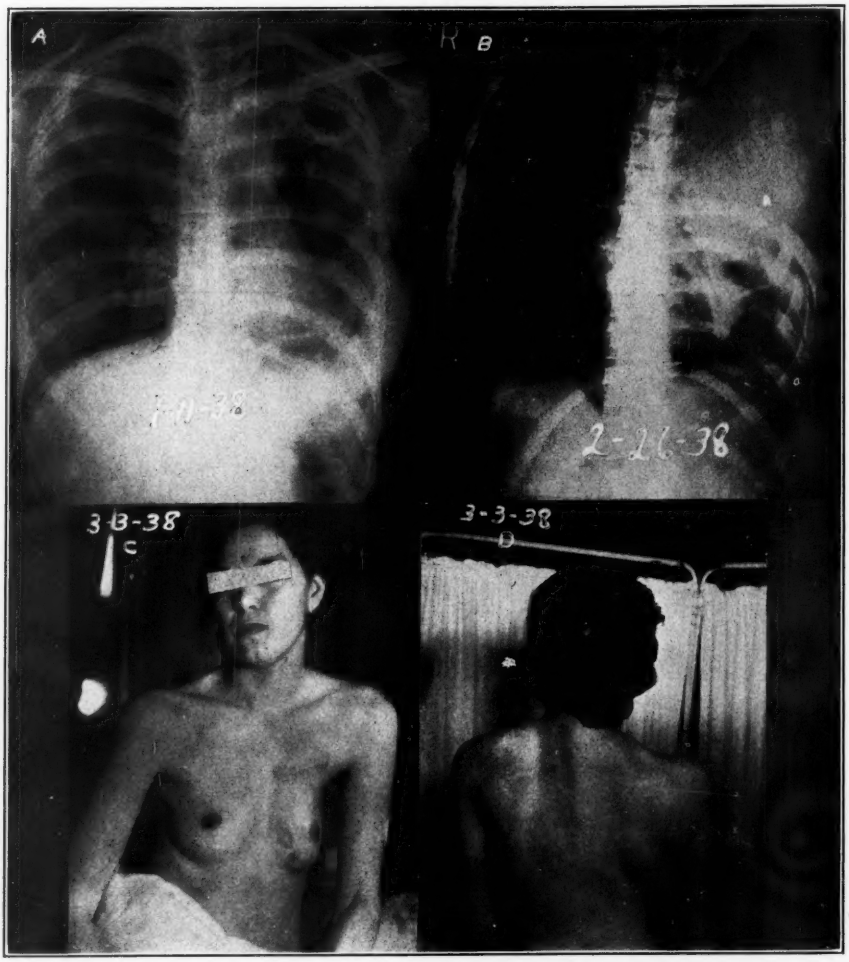

Fig. 11.—*A* and *B*, Roentgen films before and after anterior extrafascial apicolysis. The upper three ribs and cartilages have been completely excised. The fourth, fifth, sixth, and seventh ribs were cut through an oblique incision beneath the breast, tapering posteriorly at its inferior end. The silver clip denotes the summit of the lung. The photographs of the patient *(C* and *D)* show the incisions.

intercostal space. Posteriorly, it is anchored as low as the lung can be fixed without tension, usually between the fourth and fifth ribs, occasionally between the fifth and sixth. A small silver clip, employed

by neurological surgeons, is then fastened to a bit of fascia overlying the very summit of the lung, to enable determination by subsequent x-ray examinations of the position of the apex of the lung. That a satisfactory apicolysis can be achieved in this manner is apparent in Figs. 10 and 11. The trachea bifurcates usually at a level between the fifth and sixth ribs at the spine. The apex of the lower lobe can be visualized occasionally at a higher level in a postoperative roentgenogram than the silver clip on the summit of the upper lobe. Gale has emphasized the importance of fastening the lung in the depressed position.

After the apex of the lung has been brought down by apicolysis, the residual posterior portions of the upper three ribs may be removed at the same time through the anterior incision, a procedure which I have once carried out. A severe atelectasis of the unoperated lung, with some mediastinal displacement, developed thirty-six hours after operation, from which the patient died one week after the operation. I have not excised since the residual posterior segments of the upper three ribs at the time of the anterior operation. Yet in suitable cases such a one-stage thoracoplasty combined with anterior extrafascial apicolysis may prove useful.

This large extrafascial space fills with serum and air. At the subsequent upper posterior operation this space is entered and is found to be occupied by serum and coagulated fibrin in the process of organization. At the time of the anterior chondrocostectomy and apicolysis I determine the capacity of this space by pouring saline solution into the cavity, which is then promptly removed by suction and mopping with dry sponges. Depending on the extent of the apicolysis, the content of this space is found usually to vary between 125 and 275 c.c. It is apparent that no amount of rib resection will ever collapse this space completely. Downward extension of rib removal posteriorly together with excision of the transverse processes will lessen its size somewhat by lateral compression. A good portion of the space lies in the attic of the thorax, and rib removal will narrow but not obliterate its confines. It is possible, therefore, that the contents of this space may become infected subsequently. To date, however, I have not observed this complication.* I have considered the possibility of detaching a portion of the pectoralis major muscle, employing it as a pedicled flap to help fill this space, but in the interests of primary wound healing I have closed the muscle and skin tightly, depending upon organization of the serum and wound clot which accumulate to

*Since this was written I have encountered infection of the large supra-apical estrafascial space. The leaving of a "dead space" which must fill with blood and sanguineous transudate invites infection and is admittedly in conflict with the well-established surgical principle of obliterating all planes in which fluid may collect. The employment of a paraffin pack to be left in situ or the use of a synthetic rubber sponge coated with paraffin to be withdrawn at the time of the posterior operation, may eliminate this occurrence.

occupy this space. Whether an apical cavity released by apicolysis will enlarge laterally and need more extensive rib removal in a caudal direction remains to be seen.

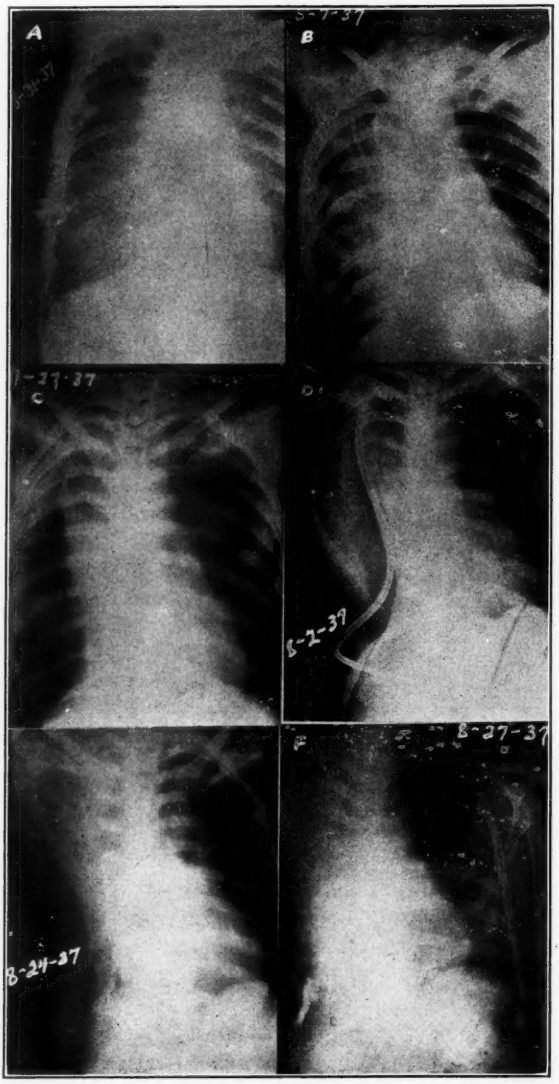

Fig. 12.—Complete costectomy for large chronic tuberculous empyema with external fistulous opening. The first film, *A*, was made on entry; the second, *B*, after packing the large pleural space with dakinized gauze. The lung has expanded slightly and an apical cavity is apparent. The remaining films (*C*, *D*, *E*, and *F*) indicate the extent of obliteration attending successive operative procedures. The first decostalization operation was done June 12, 1938, and the fourth and last, Aug. 20, 1937. The cavity is closed.

OPERATIVE REACTIONS FOLLOWING APICOLYSIS

I have the distinct impression that apicolysis augments definitely the operative reaction as contrasted with rib removal alone through short incisions. Yet, there is only one time when the apex of the lung can be depressed satisfactorily and that is at the initial operation.

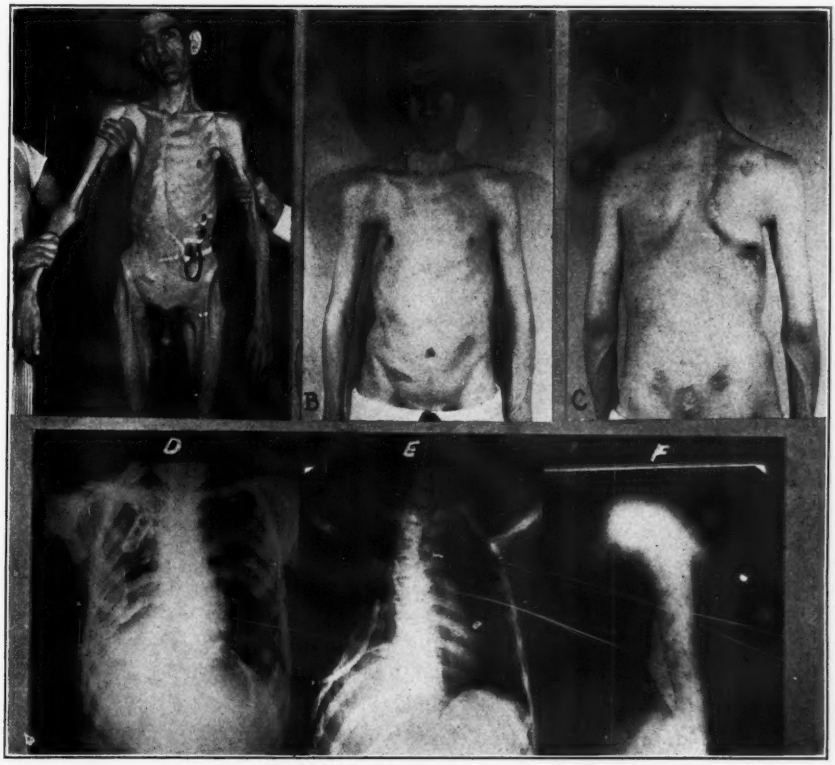

Fig. 13.—Complete costectomy for large chronic nontuberculous empyema. Following pneumonia this young man developed successively lung abscess, mediastinal abscess, esophageal fistula, and empyema with almost complete collapse of the lung. The lung could not be re-expanded, and the esophageal fistula did not close until after complete decostalization. A gastrostomy had to be done for feeding. *A*, *B*, *C*, Photographs of patient. *A* taken Aug. 13, 1937, and *B* and *C*, March 8, 1938. The date of the first decostalization operation was June 21, 1937. Evidence of rib regeneration in the anterior chest wall can be seen in *A*. The date of the last operation was Sept. 7, 1937. The patient's weight before his illness was 179 pounds. His lowest weight during the illness was 62; present weight, 130 pounds. *D*, X-ray films before decostalization (May 10, 1937). *E* and *F*. After rib removal. The drainage tube has been withdrawn, as shown in *C*, but there is still a small open sinus in the chest wall. (Oct. 13, 1937.)

Most of the increased reaction attending apicolysis concerns increased bronchial secretion and the occurrence of atelectasis. With employment of Haight's suggestion of bedside tracheal aspiration with a catheter, the hazard of such increased bronchial secretions should be

considerably lessened. Present practice in our clinic is to aspirate the trachea as the operation is completed (cyclopropane anesthesia) and again on the wards shortly after completion of the operation and later as indicated. It is indeed a very useful expedient. I have no reason to believe that atelectasis attends the anterior operation any more commonly than it does the posterior operation. It is to be admitted, however, that it is a common accompaniment of either operation, if looked for. I have not observed the occurrence of "drowned lung," a sudden massive atelectasis coming on shortly after completion of the operation, a happening which I have observed twice after a final posterior thoracoplasty.

TUBERCULOUS AND OTHER CHRONIC EMPYEMAS

In the management of large or total chronic empyema cavities, the worth of the simplified method of rib removal described here becomes most apparent. Such patients, heretofore, have been subjected by surgeons to the usual thoracoplastic method of rib excision with concomitant excision of wide sections of thickened parietal pleura, a formidable undertaking. In suitable cases the parietal pleura could be "ribboned" rather than excised by making incisions in the avascular periosteal beds after rib removal, a procedure of lesser magnitude and one which spares the innervation of the chest and abdominal walls. Experience has demonstrated, however, that large empyema cavities with thick parietal pleura can be practically obliterated by removal of the entire lengths of ribs overlying the cavity (Figs. 12 and 13). This method of dealing with ribs overlying a small empyema cavity, it is apparent, cannot be so effectual as when the cavity is extensive. A sufficiently great number of patients with large or total chronic empyema cavities has not yet been operated upon to establish exactly what the possibilities and limitations of the method are. It can be said that a cavity with a volumetric capacity of several hundred cubic centimeters can be shrunk down quickly to one holding but a few cubic centimeters. However, it would appear that a narrow paravertebral canal of variable length not uncommonly persists. Yet such small pockets can be obliterated subsequently when necessary by the usual method of excision of parietal pleura. I am hopeful that the persistence of such small empyema tracts posteriorly may be obviated at the time of the posterior operation by painting the periosteal beds with formalin solution or by reverting again to the process of "ribboning" the thick parietal pleura over short lengths posteriorly.[4]

SUMMARY

A method has been described of removing great lengths of rib subperiosteally through short incisions. Sharp periosteal raspatories and an intercostal retractor have been devised to facilitate performance

of the procedure. The writer described previously a method of removing the entire rib through short anterior and posterior incisions by taking advantage of the direction of insertion of the intercostal muscle bundles. Now, however, with employment of sharp periosteal raspatories as described herein, no heed need be taken of the direction of insertion of the intercostal muscle bundles; the rib can be freed up entirely from either direction with equal facility. If the rib has been divided at the chondrocostal junction anteriorly, it can be removed in toto through a posterior incision. If the rib is to be removed through an anterior incision, it is necessary at the preliminary posterior division of the rib to remove the short paravertebral angular segment of the rib.

This method of rib removal simplifies the operation of thoracoplasty and at the same time increases the latitude of choice of procedure for the surgeon. It has been found also that large empyema cavities may be collapsed satisfactorily without sacrifice of the thickened parietal pleura and intercostal muscle bundles by extensive costectomy.

The performance of extensive extrafascial apicolysis through an anterior incision is described, together with a method of anchoring the apex of the lung in the depressed position.

The writer wishes to express here his keen appreciation of the cordial cooperation and patience of Mr. Phelan in the work.

REFERENCES

1. Gale, Joseph W., and Midelfart, Peter A.: Extrafascial Apicolysis (Semb), Surgery **3**: 234, 1938.
2. Haight, Cameron: Intratracheal Suction in the Management of Postoperative Pulmonary Complications, Ann. Surg. **107**: 218, 1938.
3. Semb, C.: Thoracoplasty With Extrafascial Apicolysis, Acta Chir. Scandinav. **76**: 1, 1935, Supplement 37.
4. Wangensteen, Owen H.: The Pedicled Muscle Flap in the Closure of Persistent Bronchopleural Fistula, J. THORACIC SURG. **5**: 27, 1935.
5. Wangensteen, Owen H.: Complete Costectomy, Surgery **2**: 852, 1937.
6. Wangensteen, Owen H., Carlson, Herbert A., and Bowers, Warner F.: Partial Thoracoplasty for Pulmonary Tuberculosis, J. THORACIC SURG. **7**: 365, 1938.

INDICATIONS, HAZARDS, AND RESULTS OF APICOLYSIS THORACOPLASTY*

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MANY of the unsatisfactory features of the original thoracoplasty have disappeared. The more careful selection of cases, the multiple stage operation, better anesthesia, and improvements in postoperative care have all contributed to the success of the operation.

It became evident that selective, partial collapse was increasingly desirable and that its satisfactory production was difficult. In 1932 Semb began the study of this problem and in 1935[1] reported his experience with extrafascial apicolysis. Since then Coryllos[2] and Overholt[3] have published their results with the procedure.

The theoretical need for depression of the apex as well as collapse in a medial direction is obvious. The technique can be used safely by a trained surgeon, but what would be the results and hazards if the operation were used in a heterogeneous group of chronic poor risk patients? Furthermore, would such a trial help in suggesting more definite indications and limitations?

We have proceeded in an experimental manner for a period of two years, using apicolysis in a majority of our cases and applying it in 102 instances. We shall mention the surgical procedure briefly, since it has been presented elsewhere recently. The case material and the results and complications of operation will be analyzed.

TECHNIQUE OF OPERATION

Since the details of the technique have previously been described by one of us (J. W. G.),[4] only certain modifications which we have found advantageous will be given. The extent of the rib resections and lysis depends upon the patient and varies between two to four ribs. The first rib is always removed followed by increasingly shorter segments of the lower ones. This maintains a rigid support to the anterior chest wall below the level of the lysis, which lessens the reaction to the operation remarkably without sacrificing its efficiency (Figs. 1, 2, and 3). The lysis is always carried downward to the first intact rib. In our earlier cases we found that the apex would gradu-

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Read at the Twenty-First Annual Meeting of the American Association for Thoracic Surgery at Atlanta, Ga., April 4, 5, and 6, 1938.

ally rise during the first few weeks after operation. To overcome this we suture the posterior ends of the divided intercostal bundles and periosteum to the anterior edge of the neck of the first intact rib (Fig. 4). Two or three interrupted sutures between the anterior surface of the apex and these flaccid structures tuck the apex under a periosteal and muscle cap which insures fixation (Fig. 5). This procedure also eliminates any paradox which may occur at the time.

Fig. 1.—*A*, Appearance after tying the sutures. An apical cap is formed which insures against retraction of the apex to its former position. (Gale and Midelfart, Surgery 3: 239, 1938.) *B* (1) a catgut suture being placed through the posterior ends of the divided periosteum and accompanying intercostal bundles preparatory to suture through the periosteum on the anterior surface of the neck of the first intact rib; (2 and 3) the neurovascular trunk; and (4) the divided scalene muscles.

Just before the wound is closed, the divided fibers of the serratus magnus are sutured to the upper edge of the first intact rib and to the sacrospinalis as suggested by Raine.[5] This strengthens the barrier to the escape of the seroma which fills the dead space and seems to lessen the chances of fixation of the scapula.

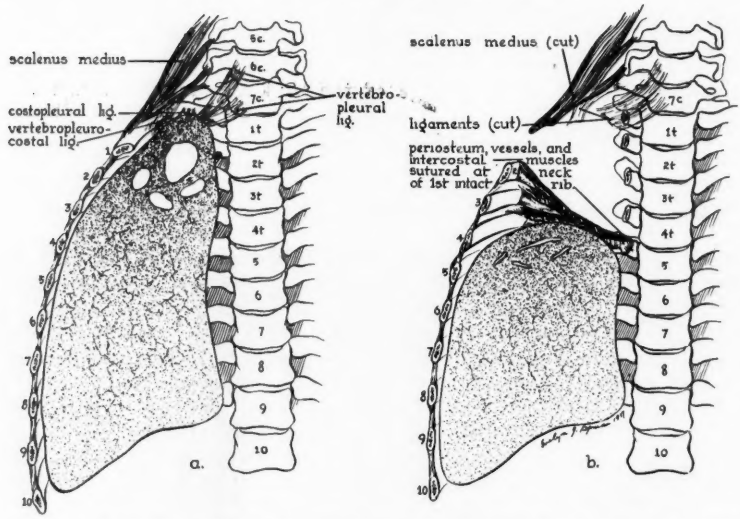

Fig. 2.—*a*, Diagrammatic sketch of the apical structures which must be divided to insure a complete apicolysis. *b*, Appearance of the lung after apicolysis and fixation of the apex in the new position. (Gale and Midelfart, Surgery 3: 240, 1938.)

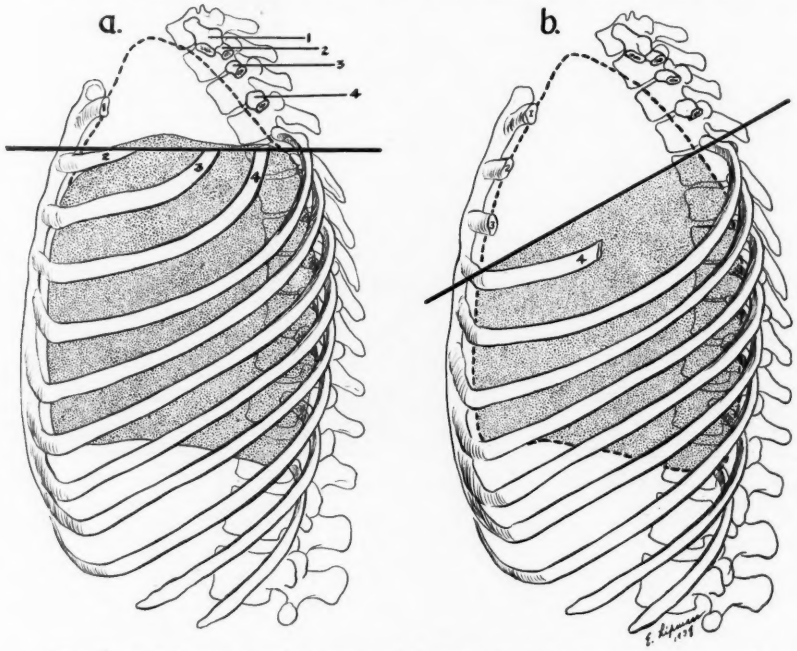

Fig. 3.—*a*, Diagram showing how graded removal of the ribs results in merely uncapping the apex, at the same time obtaining the maximum collapse of the diseased area. *b*, Result of extensive removal of the upper ribs. The support of the anterior chest wall is lost because of the oblique course of the ribs. This may result in unnecessary shock and convert a benign operation into a fatal one.

Now we always close the wound without drainage. In the earlier cases we followed the suggestion of Semb and drained for twenty-four to forty-eight hours. A study of the first sixty-five cases showed that infection occurred three times as frequently in the drained cases. Drainage also precludes the chance of obtaining the tamponade effect of the accumulated serum.

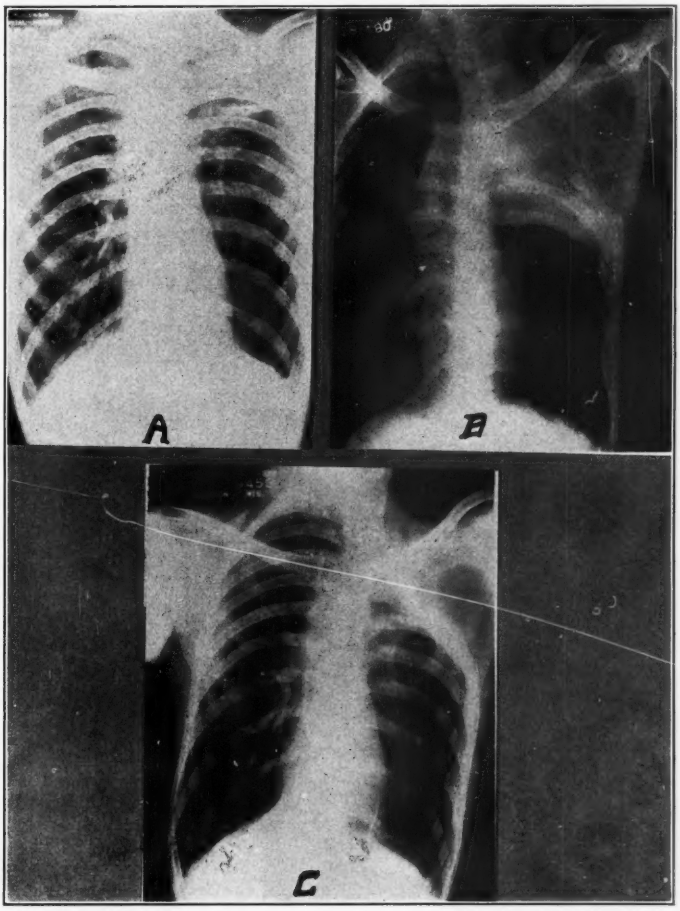

Fig. 4.—*A*, Patient with large apical cavity on the left. *B*, After two-stage thoracoplasty with apicolysis. A clip was placed at the original site of the apex and three others on the apical pleura in its lysed position. *C*, Same patient several months later. The fluid filling the dead space has been absorbed and the apex has been drawn upward due to incomplete fixation at the time of the first operation.

Since the one-stage operation is seldom adequate except in small and very high apical cavities, it is usually found necessary to perform other stages. It now seems more satisfactory to perform the anterior

stage second. This is accompanied by less shock and can be carried out at a shorter interval than could a second posterior stage. A parasternal incision is used and the anterior segments removed after separating the pectoral fibers. The seroma above the apex is seldom disturbed at this time. Within a few days anterior compression can

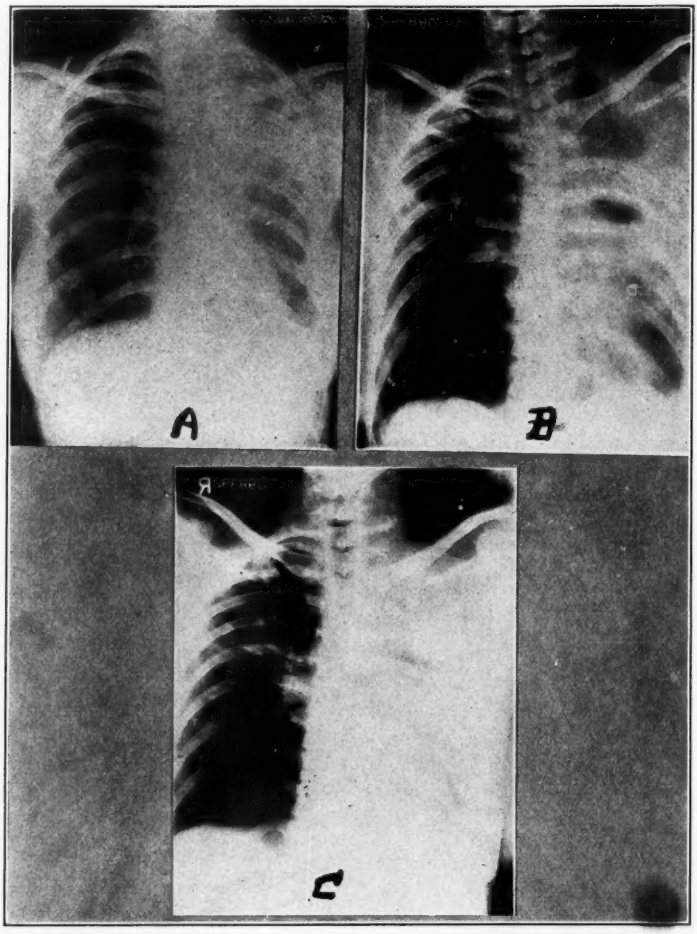

Fig. 5.—*A*, Patient with large apical cavity on the left. *B*, After first stage thoracoplasty with apicolysis. Clips show the extent of the lysis. *C*, Same patient several months later; cavity is closed. The apex has risen only about one interspace because of firm fixation at the time of the lysis.

be applied with a brace. If another posterior stage is found necessary, no hurry is indicated because the maximum amount of collapse has been obtained for the amount of rib removed at the previous operations.

Apicolysis should be applicable where the cavitation persists after an attempt at closure by paravertebral thoracoplasty. The difficulties in execution include those which are inherent in any reoperation and the distortion of anatomic relationships due to the collapse. The technique has been described.

ANESTHESIA

Anesthesia in apicolysis is more complicated than in thoracoplasty. It must be deeper, otherwise the pleural stimulation which occurs during the pleural separation will result in irregular and often violent paradoxical respiratory movement which not only adds to the difficulty of the procedure, but may be harmful to the patient.

Certain requirements have been found necessary to insure a safe and smooth anesthetic. The preanesthetic sedative of morphine and scopolamine should be gauged for the individual patient. A slow and careful induction without cough and hyperpnea will insure a smoother course throughout the operation. An open airway is imperative at all times. The use of the carbon-dioxide absorption technique allows the patient to breathe an atmosphere which is warm and moist. The anesthetic agent is supplied in an atmosphere of oxygen as similar to the air as possible but an excess of oxygen to control the cyanosis during these operations may later result in more rapid absorption and alveolar collapse. Although gases such as nitrous oxide, ethylene, and cyclopropane increase the peripheral circulation resulting in freer bleeding during the operation, this disadvantage is overshadowed by their ease of control and short recovery period which permits early cough and the evacuation of bronchial secretions.

Cyclopropane has been used throughout this series except in a few instances when we used intravenous pentothal sodium for the shorter, later stages when, after the first stage, nausea had been thought to be too prolonged.

POSTOPERATIVE COMPRESSION

We have stressed the fixation by suture of the collapsed and depressed apex, and this maneuver is really a form of compression. A series of postoperative compression measures has been used in every case. They include the individual application of posture, plastic weights, spring braces, and padded leather harnesses.

Posture is used by all patients and consists of the wedge treatment on a bolster. This position counteracts scoliosis and has a slight though inconstant immobilizing and compressing effect.

Shot and sandbags are applied early and borne easily as preliminaries to the use of other devices.

Adjustable padded spring braces can be applied for localized pressure on the upper anterior chest wall and may be used between stages.

A harness of padded leather which encircles the shoulder and is attached on the opposite hip is applied in all cases where six or more ribs have been removed. It has the advantage of producing a constant pressure with compression, immobilization, and adduction of the shoulder tissues. Forty of our cases have been fitted with this type of harness and ten with spring braces. If the apex is sutured at the time of the apicolysis there is little danger of its cephalad retraction before new rib is regenerated. We do feel, however, that the anteroposterior and lateral compression is not only helpful but imperative to obtain the maximum benefit from the operative procedure. It is to be stressed that the fitting of these braces is difficult, and the application must be forced.

INDICATIONS FOR APICOLYSIS

The ideal indications for apicolysis thoracoplasty have not been definitely stated. They would include stipulations concerning the general condition, just as for any thoracoplasty and, in addition, describe the type of lesion which would best be affected by the operation. The few authors who have published results of apicolysis have either based its use on theory of effect or have admitted their unpreparedness. It has been suggested for all bilateral cases, all giant cavities, and all cavities in the cupola.

The cases included in the present series are almost entirely foreign to any list of indications. Many were clinically active. Twenty-three of the patients were between 35 and 45 years of age, and thirteen were more than 45 years old. The average known duration of the disease was five and one-half years. There were seventeen whose disease had been present ten years or more. Many of the patients had extrapulmonary tuberculosis, some had myocardial degeneration, and some were actually cachectic.

The nontuberculous lung tissue was often severely limited in extent and defunctionalized.

The contralateral lungs were involved in 84 per cent of the cases, with an advanced lesion in 27 per cent, pathological activity by x-ray in 24 per cent, with cavitation in 15 per cent, and pneumothorax in 6 per cent.

The operated lung contained cavitation in 96 per cent of the cases, and the size of the cavity exceeded an average diameter of 6 cm. in twenty-five cases. Seventy per cent had a paralysis of the diaphragm.

About two-thirds of the patients raised more than 15 c.c. of sputum per day on admission. Eighty-one per cent of the hospital cases had a considerable fibrosis around the cavity and one-third of these were grossly fibrotic. The effects of these disadvantages will be noted later.

Nine of the 102 cases were reoperations on an area containing an uncollapsed cavity. Four of the 102 had empyema with nonexpansile lungs, and in one case, a mixed infection and a bronchopleural fistula.

PRE- AND POSTOPERATIVE CARE

There are several methods in pre- and postoperative care which seem worth emphasizing, in addition to the routine major surgical procedure. Diagnostic efforts should have identified cavitation, localized it in the third dimension, and noted the condition of the diaphragm. We agree that active contralateral disease should be controlled by pneumothorax preoperatively if the lung volume will permit. Pneumothorax has been proved impossible or ineffective on the side of operation in every case in the series. Hemoglobin levels are raised by transfusion if indicated. Patients are urged to vigorously evacuate sputum previous to their opiate. A note is given the anesthetist warning him about "wet" cases. The pre- and postoperative opiate doses have been decreased to a minimum so that consciousness and cough reflex will be least depressed. During the operation and at its conclusion, and hours afterward if necessary, a "bronchial toilet" is done with a directed catheter through the translaryngeal guide. When the patient returns to his room he is placed on his back, given six to eight liters of oxygen into the upper pharynx by nasal catheter, and roused to clear the lower air passages of sputum at short intervals. The oxygen is continued for as long after twenty-four hours as any anoxemia warrants. The thoracic service ward is fortunate in having oxygen piped to its beds from a central supply.

Every effort is made to avert shock if possible, to diagnose the occurrence, and to treat it when present. A routine rectal drip of saline solution is given on the operating table, a subcutaneous infusion within half an hour after operation, an intravenous clysis for any sign of shock, and a blood transfusion for persistent shock or a low hemoglobin level. The shock apparently is primarily due to blood volume change and to a lesser extent a nervous system or acid-base imbalance.

Atelectasis is watched for by local and general signs and symptoms and by bedside x-ray examinations. The treatment consists of the rapid application of careful, repeated, brief inhalations of concentrated carbon dioxide gas, and of bronchial aspiration. This is regularly effective when the cause of obstruction is not too dominantly exobronchial.

RESULTS OF COLLAPSE

The results in 102 cases can be presented briefly. Thirty of the operations, including sixty-one stages, were done at Statesan, and seventy-two, including one hundred and eighty-two stages, were done at the Wisconsin General Hospital during 1936 and 1937. (Apicolysis was done in 50 per cent of our thoracoplasties in 1936 and 80 per cent in 1937.) Except when especially noted, the results were similar in the two institutions. It is to be remembered that certain data on the hospital cases were obtained only a few weeks after surgery and before return of patients to the sanatoria.

The effectiveness of collapse was gauged by changes in lesion, cavity, and bronchial secretion.

The collapse of the lesion was noted to be satisfactory in 70 per cent of the cases. Thirty per cent were unsatisfactory by reason of ineffective collapse, discontinuance of surgery, or death.

In 77 per cent of the cases it was possible to complete the collapse as originally intended. Three-quarters of these were selective and localized; one-quarter was composed of intentional total collapse. In nineteen per cent of the cases the collapse was perforce partial; a total collapse became necessary in only 4 per cent.

The fate of the cavities is not always definite by x-ray examination, although the Bucky technique has been used for diagnosis in this series. In cases with cavitation 62 per cent were listed as closed, while a total of 77 per cent was called closed and indefinite. Twenty-three per cent were decreased in size but open.

It has been mentioned that many of the lesions were highly fibrotic. An analysis of the collapse results in relation to the dominant lesion type has been made. In twenty hospital cases called caseocavernous, 80 per cent had a satisfactory result; in twenty-seven cases called fibrocaseocavernous, 63 per cent were satisfactory; in nineteen cases noted to be entirely fibrotic, only 36 per cent were called satisfactory.

Large cavities (6 cm. or more in size) were closed with complete satisfaction in only 45 per cent of the cases. An analysis shows that two-thirds of these large cavities which closed were not accompanied by massive fibrosis or postoperative complications, while 90 per cent of those cavities which failed to close satisfactorily had thick walls, surrounding fibrosis, postoperative atelectasis or death, and in two cases, check valve bronchi.

The conversion of sputum from positive to negative has been a criterion of successful collapse for years. Using the results of examination of at least two sedimented sputum specimens by smear, 77 per cent of our cases were converted. We agree, however, that this method alone is incomplete, especially in cases with a marked reduction of sputum. The use of smears and guinea pig inoculations from fasting gastric contents is now routine, but only the last third of these cases was so examined. Ten per cent of the gastric-content smears from sputum-negative cases were positive; 45 per cent of the negative gastric contents were positive in animals. We must consider, therefore, the collapse effect on bronchial secretion, rather than on sputum only, and the total number of cases converted by all means was 63 per cent.

There were fourteen deaths from all causes while the patients were under our care. Sixty-four per cent occurred after first stage thoracoplasty with apicolysis; 21 per cent after revision with apicolysis; and 15 per cent after the supplementary stages.

The mortality average for first stage cases only was 9.6 per cent; for first stage and supplementary stages it was 11.8 per cent; for all apicolysis cases it was 13.7 per cent; the mortality per operation was 5.7 per cent.

The late mortality has been very low. A follow-up study of all cases shows only two additional patients dead in the past two years.

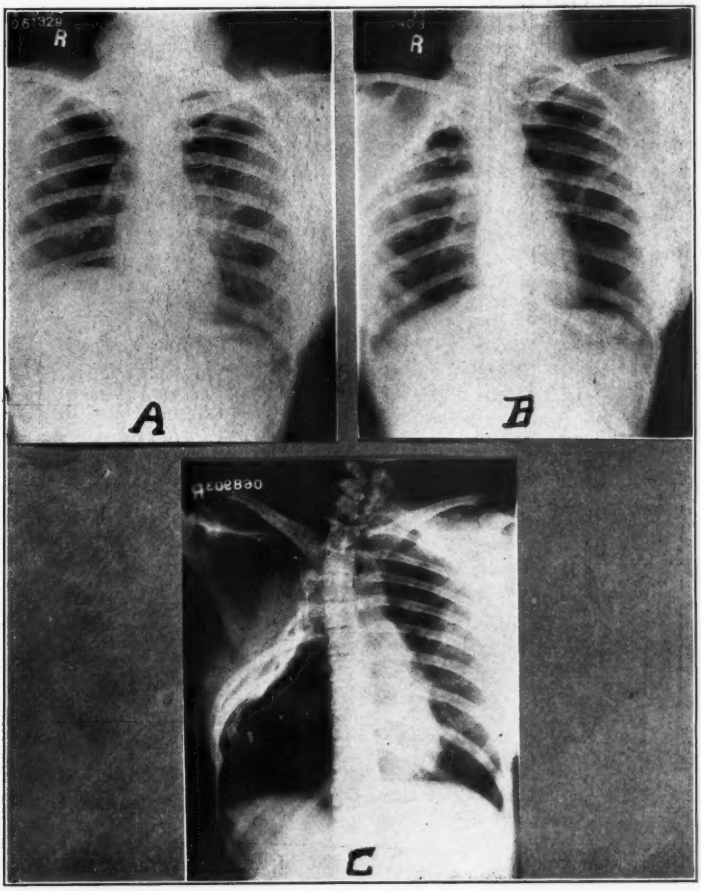

Fig. 6.—*A*, Patient with small cavity at right apex. *B*, Patient when first seen in the hospital. An inadequate thoracoplasty had failed to close the cavity. Sputum remained positive. *C*, Several weeks later following revision operation with apicolysis which succeeded in closing the cavity.

The cause in one case was coronary disease, in the other pneumonia. The total mortality is 15.6 per cent for two years.

The series is too small for a significant analysis of the causes of death, but it can be seen that they are very diverse. Some were predictable, such as the poor risk cases; others were unpredictable. Some

were tuberculous, others nontuberculous. Most of them were pulmonary, a few nonpulmonary. Some were inflammatory, and the remainder functional or mechanical. There is naturally a loose correlation of the aforementioned causes. There were six pneumonias; there were pulmonary and cerebral artery thrombosis and obstruction; there were two which resulted from cavity perforation; and toxic asthenia contributed to death in at least four cases.

Eleven apicolyses, or 15 per cent of the cases in the hospital series, were noted to be partial or small. Several of these were limited at operation, due to local factors such as fibrosis. The results show a slightly higher number of selective collapses, cavity closures, sputum conversions, and a definitely lower incidence of atelectasis and death when compared with the apicolysis series as a whole. These differences were independent of the eventual extent of collapse.

We have reconsidered the entire hospital series from the viewpoint of our present knowledge. Forty per cent, including six revisions, would be done again as routine apicolyses; 40 per cent would be done with a partial lysis (as compared with the 15 per cent actually done that way); 14 per cent would be done as a simple paravertebral operation; and 6 per cent would not be operated upon at all.

RESULTS OF REOPERATION

The results of revision, or upper stage reoperation, with apicolysis should be given. The general condition and type of resistant process in such cases need not be commented upon. Three of the nine patients died, two from complications after breaking into a cavity at operation, and one from a hemiplegia. The sputum from all six living patients is negative, although the animal inoculations from two are positive. The cavitation is closed in four, indefinite in one, and there is a persistent bronchopleurocutaneous fistula in another (Fig. 6).

RESULTS IN EMPYEMATA

There were four cases of empyema. In three, the pulmonary disease was arrested and the lung nonexpansile; the other had a bronchopleural fistula with a mixed infection. The results are not unusual. The patient with parenchymal disease (a bad risk) died on the twentieth postoperative day of a contralateral pneumonia. The other three have obtained satisfactory collapse with the apicolysis helping to decrease the space very effectively.

COMPLICATIONS OF APICOLYSIS THORACOPLASTY

The complications of importance were shock, increase of the tuberculous disease, infection of incisions, and massive atelectasis.

Shock of some degree was encountered frequently, treated prophylactically and actively, and was always transient.

Increases in the tuberculous disease occurred in fifteen cases. They were bronchogenic in most of the cases rather than reactivations.

About half interfered with a favorable end result. Eighty per cent were in the contralateral lung. In at least four cases the spread contributed to a fatal result. In all except one of the fifteen cases the patients had large quantities of sputum.

Infection of the incision played a distressing part in our thoracoplasties of all types previous to March, 1937. Although no patient in our series died with the infection present (excluding those with a fistula) the illness, delay in surgery, and prevention of collapse were serious matters. There was gross infection in 18 per cent of our cases in 1936. We abruptly took two steps to lower the incidence: drastic infectious precautions were initiated for surgical and nursing operative routine, and the use of drains and aspiration of the incision was discontinued. Drains had been used in 55 per cent of the operations in 1936 and the incidence of infection was 30 per cent, or double that of the undrained cases. The use of drains was limited to 14 per cent in 1937, and the incidence of infection was decreased to 3 per cent. The infections decreased from 18 per cent in 1936 to 9.6 per cent in 1937, including the first three months of the year, and in the hospital series the incidence fell to 4 per cent in the last nine months of 1937. Several of the chronic draining infections were basically tuberculous, but it is surprising that more were not so, since two-thirds of a series of lymph nodes which were removed from the deep tissues during operation were found to be tuberculous.

Atelectasis was the most remarkable, and serious complication was encountered. The originators of apicolysis warned that it had occurred in one-third to one-half of certain of their series, and noted that the predisposing factors included an inactive or fixed diaphragm and extensive stages. The diagnosis is not difficult but is probably often missed. The incidence in the hospital series is higher than in the sanatorium series chiefly because a bedside x-ray machine is available. The onset may be confused with prolonged shock and the later course with a tuberculous spread, but the clinical picture is distinct. It depends upon factors in the background, on localizing chest symptoms (such as cessation of expectoration, etc.), local signs (such as loss of ventilation and air content, paradoxical breathing), defunctionalizing symptoms and signs (such as anoxic anoxemia), and the x-ray appearance. It should not be mistaken for effusion, since the onset is abrupt and diffuse, the clearing is usually complete, and a pleural space before operation is only rarely present.

The incidence of massive collapse in our two series was 19 per cent. There were three times as many occurrences noted in the Hospital group as in the sanatorium group. Sixty-four per cent of the collapses occurred immediately after the apicolysis. The onset was diagnosed within the first twenty-four hours in 43 per cent, within four days in 85 per cent; 65 per cent eventually cleared, 43 per cent within one week. Of the 35 per cent which did not clear, a more

extensive successful thoracoplasty was necessitated in three cases, and four patients died with atelectasis present. (The presence of collapse was probably coincidental in two of these.) Collapse recurred one or more times in three patients and it was thought to be complicated by tuberculosis in several of the cases which did not clear.

We have correlated the occurrence of the atelectasis with several factors which seemed instrumental in its production. The condition of the diaphragm was noted to be paralyzed, or elevated and fixed, or obliterated by fibrosis in 70 per cent of all cases. Of the patients who developed atelectasis, an abnormal diaphragm was present in 90 per cent. Paralysis was present in 56 per cent of all cases; collapse occurred in 37 per cent of this group. Obliterative fibrosis was present in 14 per cent of the cases, and collapse occurred in 21 per cent of this group. Paralysis was absent in 30 per cent of the cases, and collapse occurred in only 3 per cent of this group.

Gross apico-hilar fibrosis occurred in 61 per cent of the cases in which collapse did not develop. It occurred in 93 per cent of the cases in which collapse developed.

The cases were arbitrarily classed as "wet" (with considerable sputum), or "dry" (with small amounts). The cases developing atelectasis were predominantly in the former class.

It is seen that both exo- and endobronchial factors have been present and effective in producing the atelectasis.

COMMENTS

In commenting on the foregoing sections there are many points of interest. Technically the start of mobilization must be cautious, accurate, and the progress stopped when force is necessary. Traumatization of involved lymph tissue should be avoided. A speed consistent with efficiency, hemostasis, and safety should be cultivated. The initial lysis of the apex is at times accentuated by the serum accumulation above, but the ultimate degree of collapse is well maintained at the original level of fixation, as we have determined in many cases by silver clips attached to the landmarks.

Before mentioning the results of apicolysis we would first emphasize that the material being presented for thoracoplasty has completed a cycle back to the days when only the very far-advanced case was submitted for operation. Not only are the cases increasingly poorer risks, but for the past five months we have not operated on a patient who would fall into the good risk class. This may be the result of advances in surgery which make certain cases eligible and of the modernized viewpoint of the sanatoria staffs.

We have, therefore, not found a progressive improvement in our statistical results. A fair comparison of apicolysis with the paravertebral operation cannot be made since patients subjected to the latter had less lung destruction and were better risks.

The unfavorable results include the failure to close some thick-walled and certain check valve cavities. The sputum was converted in a high percentage of the cases, but more sensitive tests of the bronchial secretion were not as completely influenced. The immediate mortality figures were probably somewhat increased by the procedure.

Atelectasis occurred and persisted too frequently, but can be avoided by noting the predisposing factors, by careful individualization, and by decreasing the size and extent of the lysis. It can be treated by taking immediate steps to evacuate and ventilate the obstructed portion of the bronchial tree.

We have not been completely successful in avoiding scoliosis, but the localization of collapse has resulted in less incarceration of the scapula.

The outstanding favorable results include a greater selectivity of collapse and conservation of functioning chest wall and lung tissue. There

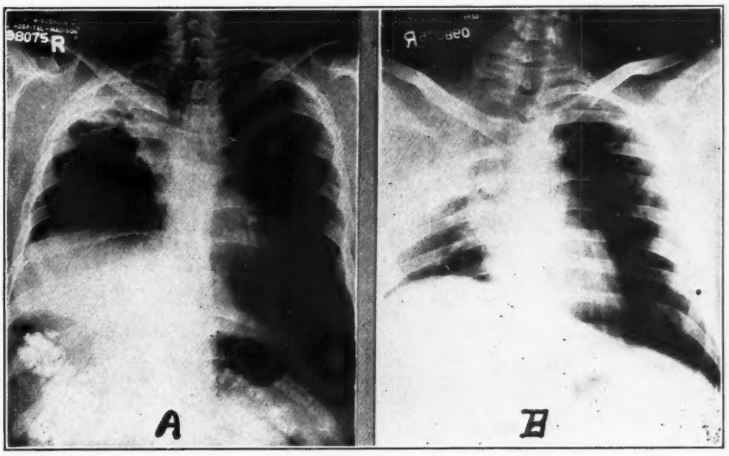

Fig. 7.—*A*, Patient with extreme fibrosis and multiple small cavities at the right apex. The right diaphragm has been paralyzed. *B*, Film taken after thoracoplasty with apicolysis. This only succeeded in displacing the fibrous apex downward en masse without appreciable effect on the size of the cavities. Apicolysis is contraindicated in this type of case.

has been a high percentage of satisfactory collapses and obliteration of cavity outlines. The collapses have been most perfect in lesions with the least fibrosis.

We have learned incidentally the control of wound infection and shock.

Probably the information of greatest value which has been derived is that concerning indications and contraindications. We now have a flexible but more definite list of criteria for the use of apicolysis. The ideal indications should include the following qualities:

1. A relatively good condition and clinical inactivity of the patient.
2. A normal, a stable, or a controlled contralateral lesion.

3. An absence of severe tuberculous or nontuberculous extrapulmonary complications.

4. A functional need for selective partial collapse therapy.

5. A thin-walled cavitative lesion of considerable size or extent.

6. Single or multiple cavities in the upper paravertebral region.

7. A bilateral apical disease which makes necessary a bilateral partial collapse.

Even more important than the indications are the contraindications. They would include:

1. A relatively poor condition and clinical activity of the patient.

2. An active, an unstable, or an uncontrolled contralateral lesion.

3. The presence of severe tuberculous or nontuberculous extrapulmonary complications.

4. Large thick-walled cavitative lesions.

5. A gross apico-hilar fibrosis, which if displaced caudally en masse might cause obstruction of larger bronchi or great vessels, or require otherwise unnecessary operations later to obtain satisfactory collapse (Fig. 7).

6. Large thick-walled cavities.

7. Dense peripleural fibrosis which prevents a safe dissection.

8. A small thin-walled cavitation occurring laterally.

CONCLUSIONS

In conclusion, apicolysis adds the definite advantage of a caudad collapse of the apex to thoracoplasty. The cases should be chosen carefully, with more limited indications, to obtain the benefit of apical collapse. Fixation of the depressed apex and postthoracoplasty compression measures contribute to a better end result.

Apicolysis adds certain definite hazards which may be minimized by obeying the contraindications, by the use of trained surgical procedure and postoperative care, and by limiting the size of the operation.

We have chosen to present the less pleasant aspects in hope that they may be a guide to the more successful use of a valuable operation.

REFERENCES

1. Semb, C.: Thoracoplasty with Extrafascial Apicolysis, Acta chir. Scandinav. **76**: 1, 1935; Holst, Johan, Semb, C., Frimann-Dahl, J.: On the Surgical Treatment of Pulmonary Tuberculosis, Acta chir. Scandinav. **76**: 1, 1935. Supplement 37.
2. Coryllos, P. N.: The Surgery of Pulmonary Tuberculosis, Quart. Bull. Sea View Hosp. **1**: 196, 1936.
3. Overholt, R. H.: Thoracoplasty with Lung Mobilization, Part I, Am. Rev. Tuberc. **34**: 411, 1937; Selective Thoracoplasty with Lung Mobilization. The Conservative Goal in Permanent Collapse Therapy, Am. Rev. Tuberc. **37**: 152, 1938.
4. Gale, J. W., and Midelfart, P. A.: Extrafascial Apicolysis (Semb), Surgery **3**: 234, 1938.
5. Raine, F.: Personal Communication.

MYOPLASTIC THORACOPLASTY

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THE "uncollapsed cavity" is a major, present-day problem in thoracoplasty for pulmonary tuberculosis. It is a challenge to all interested parties: to the internist, from the standpoint of case selection, and to the surgeon, from the standpoint of devising ways and means to accomplish a more effective collapse of lung tissue. Cavities are tridimensional; therefore, their closure requires adequate tridimensional relaxation. A perfect therapeutic pneumothorax meets just such requirements. On the other hand, the usual thoracoplasty, while assuring sufficient lateral, does not provide anteroposterior or longitudinal relaxation.

The fact that conventional or nonapicolytic thoracoplasty is productive of a high percentage of good results indicates that lateral relaxation alone is sufficient in the majority of cases. On the other hand, conventional thoracoplasty is also productive of a group of surviving patients, none the worse for their surgical experience, yet no better from the standpoint of conversion of sputum, economic independence, and public health status than they were before operation. Careful scrutiny of this small group forces one to the conclusion that most of these failures are due to a residual uncollapsed cavity and that lateral relaxation alone is insufficient in a minority of cases.

The fact that nonapicolytic thoracoplasty does not meet all requirements has long been recognized. In studying the history of the operation one is struck by the repeated efforts to incorporate the element of longitudinal relaxation into thoracoplasty itself or to evolve a substitute operation that will accomplish the desired object. These efforts date back to Tuffier's apicolysis of 1891, and from them have developed three divergent types of surgical intervention.

The first type includes all operations that can be included under the generic term "intrathoracic parietal pleurolysis." The dominant characteristics are: longitudinal relaxation from above downward; incidental lateral relaxation in the zone of surgical attack; possible anteroposterior relaxation in the same zone; a large extrapleural dead space inside the thoracic cage. The several varieties of this type are determined principally by the treatment of the dead space. From a theoretic standpoint such a space will always be a disadvantage; furthermore, foreign material, utilized for its obliteration, will constitute

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a menace. Operations of this type, while having their legitimate field of usefulness, are not included within the scope of this discussion and can be dismissed from further consideration.

The second type includes all operations designed to raise the level of the diaphragm. The dominant characteristics are: longitudinal relaxation from below upwards; no lateral or anteroposterior relaxation; loss of function, temporarily or permanently, of much good lung tissue. The varieties are determined by the manner in which the level of the diaphragm is raised and the duration of diaphragmatic paralysis. Operations of this type are likewise excluded from further consideration in this discussion.

The third type includes all operations in which the principles of thoracoplasty are combined with those of apicolysis. These operations can be grouped under the generic term "apicolytic thoracoplasty." The dominant characteristics are: longitudinal relaxation from above downward; lateral relaxation in the zone of surgical attack; anteroposterior relaxation within the same zone, dependent in degree upon the purposeful effort to secure it; a dead space outside the thoracic cage. The several varieties are determined by the anatomical planes within which the apical dissection is accomplished, the surgical treatment of the chest wall attachments to sternum and vertebral column, the treatment of the resulting dead space and the efforts made to prevent a postoperative rise of the lowered apex. From a theoretic standpoint operations of this type present disadvantages, chief of which are: a dead space immediately adjacent to the superior mediastinum, extensive dissection of pleura from mediastinal structures, trauma to lymphatics which may contain tubercle bacilli, the difficulty of anchoring the apex in its new position, concealed paradoxical respiration. The present discussion is concerned with one variety of apicolytic thoracoplasty.

The reader is referred to Alexander[1] for a complete discussion of the history and evolution of thoracoplasty. Apparently the first apicolytic thoracoplasty was performed by Friedrich in 1908. Other surgeons, notably Bull, Casper, and Lilienthal, have elaborated upon it, employing a preliminary intrathoracic, extrapleural dissection. Semb[6] has made an important contribution by utilizing an extrafascial dissection and by demonstrating the ease with which the parietal pleura can be separated from all cervical, vertebral, and sternal attachments. This obviates all intrathoracic dead space but does not anchor the apex or overcome the other disadvantages of apicolysis. The operation to be described employs extrafascial dissection and attempts to meet the resultant problems.

OPERATIVE TECHNIQUE

The patient is placed in lateral decubitus, with thoracoplasty supports behind him and table tilted slightly backward. The side for

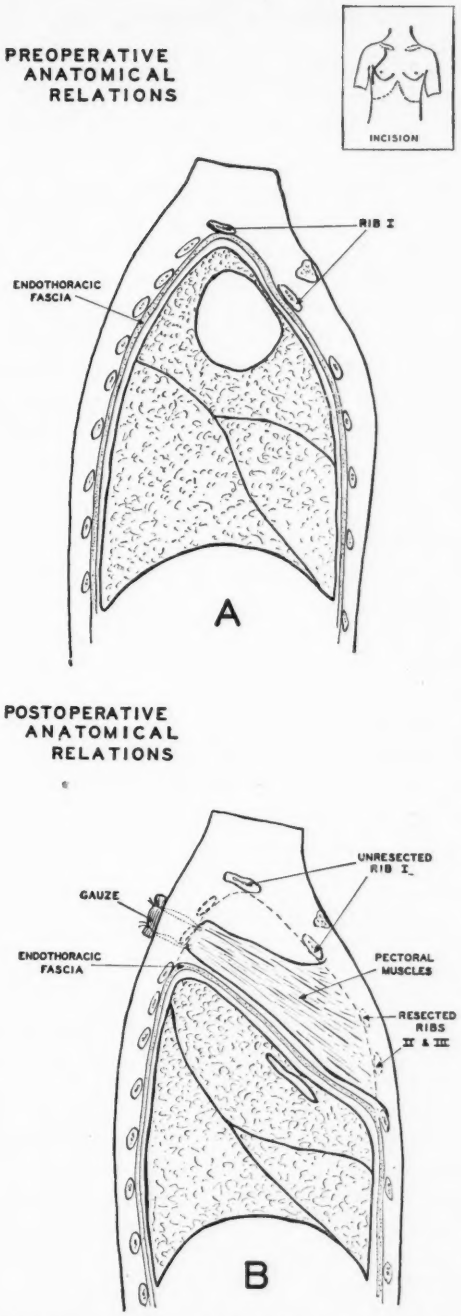

Fig. 1.—*A*, Preoperative anatomic relationships (inset shows line of incision on anterolateral chest wall). *B*, Postoperative anatomic relationships, showing extrafascial dissection of apex and transplantation of pectoral muscles into their new positions.

operation being uppermost, that arm is abducted 100° and there secured. In the skin preparation, axilla, anterior chest wall, posterior chest wall, and arm to elbow are all included, the patient's back finally resting against sterile pads and towels.

The skin incision starts at the mid-clavicle, circles downward across the inferior border of the pectoral muscle to the mid-axilla, and thence continues caudad in the mid-axillary line to the level of the fifth or sixth rib (Fig. 1). This incision should avoid the thin, hair-bearing axillary skin. The skin and subcutaneous tissue are quickly brushed away, medially and laterally, from the pectoralis major. This muscle is split, as in a radical breast operation, into its sternal and clavicular portions, care being taken, however, to avoid injury to the neurovascular pedicle. The sternal fibers are freed laterally to their insertion into the humerus and are there divided, allowing the muscle, with blood and nerve supply intact, to be reflected medially. The pectoralis minor is freed and divided at its insertion into the coracoid, care being again exercised to avoid injury to the nerve and blood supply. This rapidly affords, with very little bleeding, a wide exposure of the anterolateral chest wall between the levels of the first and fourth ribs. The second and third ribs, with their costal cartilages, are subperiosteally resected from the sternum to the scapular line. The first rib is not disturbed. The first intercostal muscles are sectioned close to the edge of the first rib and through this opening an extrafascial apicolytic dissection is easily initiated. Excellent visibility is obtained. As the dissection progresses it becomes necessary to section the first intercostal muscles, the second periosteal sheath, the second intercostal muscles, and the third periosteal sheath at the stumps of the two resected ribs, so as to expose, from within the thoracic cavity, the remaining portions of those ribs. Their resection is then completed back to the vertebral bodies. The extrafascial dissection is continued downward from the anterior end of the first rib, parallel to the sternum but lateral to the internal mammary vessels. The apex may be dropped to any desired level within the operative field.

After the apical dissection has been completed and hemostasis has been satisfactorily secured, the free ends of the two pectoral muscles are transplanted across the lowered apex into the first, second, or third interspace, as desired (Fig. 1). Doubled chromic catgut mattress-sutures are utilized on long straight bayonet needles, passed from within the thoracic cavity outward through the previously prepared posterior chest wall. There should be sufficient needles so that each may be discarded after one such passage. The sutures are tied externally at their points of emergence from the posterior chest wall. The operative wound is then closed without drainage after quilting the skin flaps down against the underlying soft tissues.

DISCUSSION OF OPERATIVE TECHNIQUE

The important features of the foregoing operation are: the anterolateral approach; the complete resection of ribs from sternum to vertebrae; the extrafascial dissection of the apex; the anchorage of the apex; the prevention of paradoxical motion; and the obliteration of the dead space. Each of these features will be separately considered; the relation of this type of operation to the whole thoracoplasty program will then be discussed.

There is nothing new in the anterolateral approach. Alexander[2] has reviewed the subject of anterior operations, and the reader is again referred to him for an historical résumé. Ecot and Jullien[5] have pointed out advantages which, in their opinion, make it a desirable first stage approach in every thoracoplasty program. No previously described incision has extended to the clavicle. The outstanding advantages of the incision as described are: a relatively bloodless field; a very rapid and simple approach to the many anatomic structures involved in the operation; and the excellent visual control of the entire operative field.

Anterior relaxation requires the resection of ribs and costal cartilages to the sternum. This is accomplished with the utmost ease through the approach described. Even more significant is the fact that complete resection from sternum to vertebral body is entirely feasible. Manifestly, the complete resection of the second and third ribs, with or without the resection of the first rib, is sufficient for one stage. The first rib may be ignored without prejudice to the thoracoplastic collapse because all chest wall and pleural structures will have been separated from it, dropped to a lower level, and prevented from reuniting with it by the transplanted pectorals. It will not, therefore, influence the subsequent behavior of the cavity. The advantages of ignoring the first rib are: shortening of operating time; avoidance of risks connected with its resection; stabilizing effect in prevention of scoliosis. The possible disadvantages of leaving the first rib in situ are the obstruction to firm embedding of the scapula and maintenance of an undesirable dead space immediately below the rib.

The extrafascial dissection of the apex conforms to the technique described by Semb, except that the dissection is initiated anteriorly and carried toward the posterior chest wall. The anterior approach carries this added advantage: the chest wall attachments to the sternum can be sectioned under a direct visual control that minimizes the risk of concealed or uncontrollable hemorrhage.

One maneuver accomplishes the anchorage of the apex, the prevention of paradoxical respiration, and the filling of the dead space with viable tissue. The moment the mobilized pectorals are sutured to the posterior chest wall these three objects are accomplished. The pectoralis minor should be first implanted at the lowermost desired level.

The pectoralis major is implanted across the minor at a slightly higher level. Both of these muscles are long enough, if sectioned at their points of insertion into coracoid and humerus, to comfortably reach the posterior chest wall. There has been no opportunity to observe, at autopsy, the fate of the transplanted muscles, but exploration of two sinuses has shown that the muscles have remained in their transplanted positions, and that they have been well nourished.

The availability of the pectoral muscles for filling a dead space or for maintaining relaxation gained by other methods has long been recognized. Tuffier[8] and Archibald[4] have used them as a muscle filling in extrapleural apicolysis. Alexander[3] has employed them for subcostal compression. Both Shivers[7] and Archibald[4] have transplanted the tendons of the minor and major pectorals into the posterior stump of the trapezius as part of the upper stage of the classical Sauerbruch paravertebral thoracoplasty.

The operation as described assures longitudinal relaxation and it offers a great degree of anteroposterior relaxation in the zone of surgical attack. It does not provide any lateral relaxation below the level of the third rib; therefore, it must be augmented by additional posterolateral thoracoplasty stages.

In theory, this operation should constitute the first stage of any thoracoplasty program in which it is to be utilized. Virginal anatomic relationships render the apicolytic dissection easier and safer. This dissection and the pectoral transplantation should be done coincidentally. In practice the operation as described can be done and has been done as the second stage of a thoracoplasty program. The results have not been gratifying.

CASE REPORTS

The brief histories of ten patients subjected to this operation in the New York State District Tuberculosis Hospitals during the calendar year 1937 are given as follows. All these patients were suffering from moderately advanced or far-advanced pulmonary tuberculosis. Other collapse measures had failed. In each case the decision for thoracoplasty was arrived at in staff or consultation conference after a thorough presurgical study had shown that the patient was a suitable candidate for such a procedure. In each case an apicolytic operation was considered necessary to assure cavity closure.

CASE 1.—F. B. (B. M. H.),* No. 1211, aged 41 years, female, white, single, had pulmonary tuberculosis of two years' duration, bilateral, maximum disease, right. Previous treatment: three months' bed rest; unsuccessful right pneumothorax. Preoperative x-ray showed dense infiltration of right lung from rib three to apex with a cavity 6 by 4 cm. and light infiltration, rib three to rib five; the left lung had multiple areas of granular infiltration. There were no other tuberculous foci.

Stage 1.—May 19, 1937. Avertin 70 mg., nitrous oxide. Retroinfrascapular incision. Resection of 16 cm. of rib three, 15 cm. of rib two. Operation terminated

*Hermann M. Biggs Memorial Hospital, Ithaca, N. Y.

because of fall in blood pressure. Postoperative course: mild shock; maximum temperature 103° F. on fifth day; fever for seventeen days.

Stage 2.—June 15, 1937. Avertin 80 mg., nitrous oxide. (Apicolytic and myoplastic stage.) Anterolateral incision as described in text. Resection of 4 cm. of rib one, 14 cm. of rib four. Apex easily freed and dropped to rib four. Pectorales major and minor sutured to posterior chest wall. Postoperative course: no shock; maximum temperature 101.8° F. on second day; fever for seven days.

Stage 3.—July 7, 1937. Avertin 85 mg., nitrous oxide. Retroinfrascapular incision reopened. Resection of 11 cm. of rib seven, 12.5 cm. of rib six, 13 cm. of rib five, 2 cm. of rib four. Postoperative course: moderate shock on first day; maximum temperature 101.4° F. on second day; slight fever for two months.

Present Status.—No evidence of cavity on x-ray film. Sputum: weight not recorded (patient may swallow her sputum); negative on concentration six months. Wounds: anterior, tuberculous sinus lying between pectorals and rib one; posterior, well healed. Opposite lung: increased disease after thoracoplasty; lesion now regressive; no cavity. Slight scoliosis. Arm function: lateral abduction 100°. Patient still under surgical dressing care.

Result.—Unsuccessful.

CASE 2.—M. H. (H. F. T. H.),* No. 53, aged 27 years, female, white, single, had pulmonary tuberculosis of three years' duration, bilateral, maximum disease, left. Previous treatment: sanatorium care; left pneumothorax, two years, ineffectual pneumonolysis; pneumothorax abandoned eight months before operation. Preoperative x-ray film showed left lung with infiltration throughout; cavity, upper lobe, 2.7 by 4 cm.; second cavity, apex of lower lobe, 5 by 6.5 cm.; right lung, infiltration from apex to rib five; questionable cavity within circle of rib one. Renal tuberculosis.

Stage 1.—May 3, 1937. Avertin 70 mg., nitrous oxide. (Apicolytic stage.) Retroinfrascapular incision. Resection of 23 cm. of rib two, 17 cm. of rib one. Apex easily freed and dropped to rib three posteriorly. Postoperative course: uneventful.

Stage 2.—May 24, 1937. Avertin 70 mg., nitrous oxide. (Apicolytic stage.) Retroinfrascapular incision reopened. Resection of 25 cm. of rib three, 27 cm. of rib four. Apex further dropped to rib four. Postoperative course: slight fever for twenty-nine days, otherwise uneventful.

Stage 3.—June 25, 1937. Avertin 70 mg., nitrous oxide. (Myoplastic stage.) Anterolateral incision as described in text. Resection of 30 cm. of rib five. Pectoralis major sutured to posterior chest wall; pectoralis minor transplanted anterior to major; anterior edge of serratus approximated to lateral border of pectorals. Postoperative course: staphylococcal infection on eighth day; slight drainage for thirty-three days.

Stage 4.—Aug. 13, 1937. Avertin 70 mg., procaine 1 per cent. Retroinfrascapular incision reopened. Resection of 22 cm. of rib six, 2.5 cm. of rib five, 20 cm. of rib seven, 19 cm. of rib eight. Postoperative course: uneventful.

Stage 5.—Sept. 17, 1937. Avertin 70 mg., procaine 1 per cent. Lower half of previous incision reopened and extended downward and forward. Resection of 20 cm. of rib nine, 17 cm. of rib ten, 10 cm. of rib 11. Postoperative course: uneventful.

Present Status.—Anterior cavity closed on x-ray examination; questionable x-ray evidence of posterior cavity. Sputum: amount and bacillary content unchanged. All wounds well healed. Opposite lung: slight increase in activity, probable source of bacilli in sputum; no cavity; bronchial tuberculosis. Moderate upper dorsal scoliosis. Arm function: lateral abduction 90°; anterior adduction in horizontal plane, 60 per cent of normal. Vital capacity: before, 1550 c.c., after, 1300 c.c.; 16.1 per cent loss. Patient still in hospital under medical supervision.

Result.—Unsuccessful.

*Homer Folks Tuberculosis Hospital, Oneonta, N. Y.

CASE 3.—C. C. (B. M. H.), No. 261, aged 38 years, female, white, married, had pulmonary tuberculosis of twelve years' duration, bilateral, maximum disease, left. Previous treatment: sanatorium care, one year; intermittent bed rest at home. Preoperative x-ray film showed left lung with dense fibrosis at apex; cavity 5 by 5 cm.; fibrous and calcified foci in second intercostal space; right lung, small calcified areas throughout. No other tuberculous foci.

Stage 1.—July 6, 1937. Avertin 70 mg., nitrous oxide. (Apicolytic and myoplastic stage.) Anterolateral incision as described in text. Resection of 19 cm. of rib two, 29 cm. of rib three. Apex freed with difficulty, especially in spinal gutter, and dropped to rib four posteriorly. Pectorales minor and major sutured to posterior chest wall. Postoperative course: no shock; temperature 103.6° F. on fifth day, slight fever for one month; drainage from wound, two months.

Stage 2.— Aug. 17, 1937. Avertin 70 mg., procaine 1 per cent. Retroinfrascapular incision. Resection of 19 cm. of rib four, 18.5 cm. of rib five, 3 cm. stump of rib two. Postoperative course: no shock; maximum temperature 101° F. on third day; slight fever for twenty days.

Stage 3.—Sept. 15, 1937. Avertin 75 mg., procaine 1 per cent. Retroinfrascapular incision reopened and extended. Resection of 22 cm. of rib six, 18 cm. of rib seven, 7 cm. of rib five, and some regenerated bone. Postoperative course: no shock; maximum temperature 101° F. on third day; slight fever for twelve days.

Present Status.—X-ray film showed uncollapsed cavity, 1.2 by 1.5 cm., at apex, posteriorly. Sputum: average weight before operation 30 gm., after, 10 gm.; "few" bacilli on direct smear. All wounds well healed. Opposite lung unaffected by operation. Moderate scoliosis. Arm function: lateral abduction 95°; anterior adduction in horizontal plane, 50 per cent of normal. Patient still in hospital under medical supervision.

Result.—Unsuccessful.

CASE 4.—B. M. (B. M. H.), No. 1699, aged 38 years, female, white, married, had pulmonary tuberculosis of twenty-one months' duration, bilateral, maximum disease, left. Previous treatment: diagnosis at six months; bed rest four months; unsuccessful left pneumothorax; left phrenic nerve crushing; recrushing five months before admission; diaphragm excursion two cm. on admission. Preoperative x-ray film showed left lung with dense infiltration apex to rib three; cavity 3.5 by 4 cm.; second smaller cavity; right lung, fibrous infiltration in first intercostal space. No other tuberculous foci.

Stage 1.—June 15, 1937. Procaine 1 per cent. Retroinfrascapular incision. Resection of 11 cm. of rib six, 12 cm. of rib five, 14 cm. of rib four, 12 cm. of rib three, 7 cm. of rib two. (This patient was regarded as a very poor risk; the first stage was undertaken as a trial operation. She responded better than anticipated and the apicolytic operation was later performed.) Postoperative course: no shock; maximum temperature 101.6° F. on seventh day.

Stage 2.—July 7, 1937. Procaine 1 per cent. (Apicolytic and myoplastic stage.) Anterolateral incision as described in text. Resection of 15 cm. of rib two, 15 cm. of rib three. Apex easily freed and dropped to rib four. Pectorales minor and major sutured to posterior chest wall across apex. Postoperative course: mild shock (95 systolic); maximum temperature 101.4° F. on second day, moderate fever for thirteen days.

Stage 3.—Aug. 18, 1937. Avertin 70 mg., procaine 1 per cent, nitrous oxide. Retroinfrascapular incision reopened. Resection of 15 cm. of rib seven, regenerated portions of ribs six, five, four. Postoperative course: no shock; fever persisted until drainage of abscess of anterior wound on Sept. 14, 1937; sinus has persisted since.

Present Status.—No evidence of cavity on x-ray examination. Sputum: average weight before operation 46 gm., after, 2 gm.; negative on concentration six months (positive culture twice during that time). Wounds: anterior, tuberculous sinus which is very slowly improving; posterior, tuberculous sinus. Opposite lung unaffected by operation. No scoliosis. Arm function: lateral abduction 90°; anterior adduction in horizontal plane greatly restricted. Patient still under surgical dressing care.

Result.—Unsuccessful.

CASE 5.—M. G. (H. F. T. H.), No. 3155, aged 39 years, male, white, single, had pulmonary tuberculosis of fifteen years' duration, bilateral, maximum pulmonary disease and empyema, mixed, with bronchopleural fistula, right. Previous treatment: few pleural aspirations at onset; neglected case for fifteen years; open thoracotomy two months ago. Preoperative x-ray film showed right lung, 60 per cent collapsed by empyema, apex adherent and honeycombed, cavity 3 cm. in diameter; mediastinal shift to right; left lung, nodular infiltration throughout. No other tuberculous foci. Congo red test: 45 per cent absorption in one hour.

Stage 1.—June 22, 1937. Avertin 70 mg., nitrous oxide. (Apicolytic stage.) Retroinfrascapular incision. Resection of 18 cm. of rib four, 16 cm. of rib three, 13 cm. of rib two. Apex incompletely freed with great difficulty and dropped only 3 cm. Upper portion of rhomboids and lower end of scalenus medius sutured into space beneath rib one; lower portion of rhomboids folded over transverse processes and there sutured. Postoperative course: no shock; temperature above 100° F. for eleven days; drainage from upper angle of wound on eleventh day, persisting seven days.

Stage 2.—July 14, 1937. Avertin 70 mg., nitrous oxide. (Apicolytic and myoplastic stage.) Anterolateral incision as described in text. Resection of 8 cm. of rib two, 13 cm. of rib three, 16 cm. of rib four. Another effort with poor success to free apex anteriorly. Pectorales major and minor sutured, across apex, to previously infolded mass of rhomboid and scalene muscles. Postoperative course: profuse drainage from upper third of anterior wound on ninth day, persisting until death four months later.

Stage 3.—Sept. 22, 1937. Avertin 70 mg., procaine 1 per cent, nitrous oxide. Lower two-thirds of posterior incision reopened. Resection of 20 cm. of rib five, 21.5 cm. of rib six, 20 cm. of rib seven, 18 cm. of rib eight. Postoperative course: uneventful.

Stage 4.—Oct. 18, 1937. Avertin 70 mg., procaine 1 per cent. Posterior incision from rib nine to rib twelve. Thoracotomy tract accidentally entered and operation terminated. Postoperative course: fever to 101.5° F.; wound infection on fourth day; death on nineteenth day from acute spread of tuberculosis to opposite lung. Autopsy requested but not secured.

Result.—Died.

CASE 6.—A. S. (H. F. T. H.), No. 3145, aged 23 years, male, white, married, had pulmonary tuberculosis of two years' duration, bilateral, maximum disease, left. Previous treatment: sanatorium care three months; unsuccessful pneumothorax left. Preoperative x-ray film showed left lung, fibrotic infiltration above rib six, multiple cavities, anterior and posterior, from 2 cm. to 3 by 5.5 cm.; right lung, fibrotic infiltration above rib three. No other tuberculous foci.

Stage 1.—June 22, 1937. Avertin 70 mg., nitrous oxide. (Apicolytic stage.) Retroinfrascapular incision. Resection of 16 cm. of rib four, 15 cm. of rib three, 12 cm. of rib two. Apex freed with difficulty and dropped to rib four. Rhomboids and upper portion of serratus anterior sutured into dead space. Postoperative course: no shock; separation of upper half of wound requiring dressings for forty days.

Stage 2.—July 11, 1937. Avertin 70 mg., nitrous oxide. (Myoplastic stage.) Anterolateral incision as described in text. Resection of 9 cm. of rib two, 2.5 cm. of rib three, 14 cm. of rib four. Apical dissection could not be completed because of scar tissue resulting from Stage 1. Pectorales major and minor sutured to previously infolded rhomboids at levels of ribs two, three, four. Postoperative course: no shock; wound infection and drainage for sixty days.

Stage 3.—Sept. 17, 1937. Avertin 70 mg., nitrous oxide. New retroinfrascapular incision. Resection of 25 cm. of rib five, 22.5 cm. of rib six, 17.5 cm. of rib seven. Postoperative course: no shock; temperature to 104.2° F., above 100° F. for twelve days; mild wound infection.

Present Status. No evidence of cavity on x-ray examination. Sputum: average weight before operation 60 gm., after, 8 gm.; negative on concentration three months, negative on culture one month. All wounds well healed. Opposite lung unaffected by operation. No scoliosis. Arm function: lateral abduction 80°; anterior adduction in horizontal plane 50 per cent of normal. Vital capacity: before, 2200 c.c., after, 1800 c.c.; 18.2 per cent loss. Patient at home; left hospital against advice for socioeconomic reasons.

Result: Apparently successful.

CASE 7.—H. P. (H. F. T. H.), No. 3184, aged 27 years, male, white, single, had pulmonary tuberculosis of six years' duration, unilateral, right. Previous treatment: sanatorium care three years; unsuccessful right pneumothorax; temporary phrenic nerve crushing one year before admission. Preoperative x-ray films showed right lung with dense infiltration above rib four; cavity 1 by 2 cm. at apex; left lung, healed lesion extreme apex. No other tuberculous foci.

Stage 1.—June 25, 1937. Avertin 70 mg., nitrous oxide. (Apicolytic and myoplastic stage.) Anterolateral incision as described in text. Resection of 15 cm. of rib two, 20 cm. of rib three. Apex easily freed and dropped to rib four posteriorly. Pectoralis minor discarded; pectoralis major sutured to posterior chest wall across apex. Postoperative course: moderate shock six hours after operation, easily relieved by simple measures; maximum temperature 102.4° F. on first day, above 100° F. for six days; serum pocket below right clavicle drained for seventy days.

Stage 2.—July 15, 1937. Procaine 1 per cent, nitrous oxide. Retroinfrascapular incision. Resection of 9 cm. of rib three, 17 cm. of rib four, 4 cm. of rib two. Postoperative course: uneventful.

Present Status.—No evidence of cavity on x-ray examination. Sputum: average weight before operation 5 gm., after, 3 gm.; negative on concentration nine months, negative on culture six months. All wounds well healed. Opposite lung unaffected by operation. No scoliosis. Arm function: lateral abduction unrestricted; anterior adduction in horizontal plane 90 per cent of normal. Vital capacity: before, 3600 c.c., after, 3050 c.c.; 15.3 per cent loss. Patient at home, at light work.

Result: Apparently successful.

CASE 8.—M. B. (M. M. T. H.)* No. 4093, aged 23 years, female, white, single. Pulmonary tuberculosis of two years' duration, bilateral, maximum disease, right, therapeutic left pneumothorax. Previous treatment: sanatorium care since onset; unsuccessful right pneumothorax at three months; therapeutic left pneumothorax at nine months. Preoperative x-ray films showed right lung with a cavity 7 cm. diameter at apex; left lung, 25 per cent collapsed by therapeutic pneumothorax. No other tuberculous foci.

*Mt. Morris Tuberculosis Hospital, Mt. Morris, N. Y.

Stage 1.—Sept. 6, 1937. Avertin 70 mg., nitrous oxide. (Apicolytic and myoplastic stage.) Anterolateral incision as described in text. Resection of 15 cm. of rib two, 20 cm. of rib three. Apex easily freed and dropped to rib four posteriorly. Pectorals sutured across apex into third interspace posteriorly. Postoperative course: no shock; retained bronchial secretions requiring bedside bronchoscopy; maximum temperature 105° F. on third day, before bronchoscopy, 100.5° F. after bronchoscopy; otherwise uneventful and wound healing per primam until fourteenth day when contralateral spontaneous pneumothorax and patient's death occurred. Autopsy requested but not secured.

Result: Died.

CASE 9.—H. M. (H. F. T. H.) No. 3142, aged 34 years, female, white, married, had pulmonary tuberculosis of eleven years' duration, bilateral, maximum disease, left. Previous treatment: sanatorium care two years; unsuccessful left pneumothorax. Preoperative x-ray film showed left lung, moderate infiltration throughout; cavity 4 by 5 cm., apex upper lobe; cavity, same size, apex lower lobe; right lung, slight infiltration above rib three. No other tuberculous foci.

Stage 1.—June 21, 1937. Avertin 70 mg., nitrous oxide. (Apicolytic stage.) Retroinfrascapular incision. Resection of 15 cm. of rib three, 12 cm. of rib two, 15 cm. of rib four, 16 cm. of rib five. Apex easily freed and dropped. Rhomboids folded over transverse processes into spinal gutter. Postoperative course: slight shock at end of operation; maximum temperature 103.4° F. on sixth day; drainage from upper angle of wound on sixth day, persisting for forty days.

Stage 2.—Sept. 21, 1937. Avertin 70 mg., nitrous oxide. (Myoplastic stage.) Anterolateral incision as described in text. Resection of 9.5 cm. of rib two, 14 cm. of rib three, 8.5 cm. of rib one. Impossible to free apex anteriorly because of dense scar tissue. Pectorals transplanted under periosteal sheath of rib one. Postoperative course: uneventful.

Stage 3.—Oct. 14, 1937. Avertin 70 mg., procaine 1 per cent. New retroinfrascapular incision. Resection of 16 cm. of rib six, 17 cm. of rib seven, 9.5 cm. of regenerated rib five. Postoperative course: mild infection at lower angle of wound and drainage for fifty days.

Present Status.—No evidence of cavity on x-ray examination. Sputum: average weight before operation 175 gm., after, 10 gm.; negative on concentration five months. All wounds well healed. Opposite lung unaffected by operation. No scoliosis. Arm function: lateral abduction 90°; anterior adduction in horizontal plane 50 per cent of normal. Vital capacity: before, 1350 c.c., after, 1150 c.c.; 14.8 per cent loss. Patient discharged from hospital to sanatorium near home for continuation of medical care.

Result: Apparently successful.

CASE 10.—B. C. (H. F. T. H.), No. 4925, aged 24 years, male, white, married, had pulmonary tuberculosis of nine years' duration, unilateral, right. Previous treatment: undiagnosed three years; sanatorium care past sixteen months; unsuccessful right pneumothorax. Preoperative x-ray examination showed right lung, moderate infiltration throughout; cavity 6 by 6.5 cm. near apex; left lung, healed lesion first intercostal space. No other tuberculous foci (Fig. 2*A*).

Stage 1.—Oct. 12, 1937. Avertin 70 mg., nitrous oxide. (Apicolytic and myoplastic stage.) Anterolateral incision as described in text. Resection of 22 cm. of rib two, 27 cm. of rib three. Apex freed with difficulty and dropped to rib five posteriorly. Pectoralis minor sutured over apex to third and fourth interspaces posteriorly; pectoralis major sutured over minor to second and third interspaces

posteriorly. Postoperative course: no shock; maximum temperature 102° F. on third day, fever above 100° F. for seven days; slight increase in disease in opposite lung; postponement of stage two for four months (Fig. 2 *B*).

Stage 2.—Feb. 4, 1938. Avertin 70 mg., nitrous oxide. Retroinfrascapular incision. Resection of 25 cm. of rib five, 23 cm. of rib four, 25 cm. of rib six. Postoperative course: uneventful.

Present Status.—No evidence of cavity on x-ray examination. Sputum: average weight before operation 90 gm., after, 60 gm.; negative on concentration three

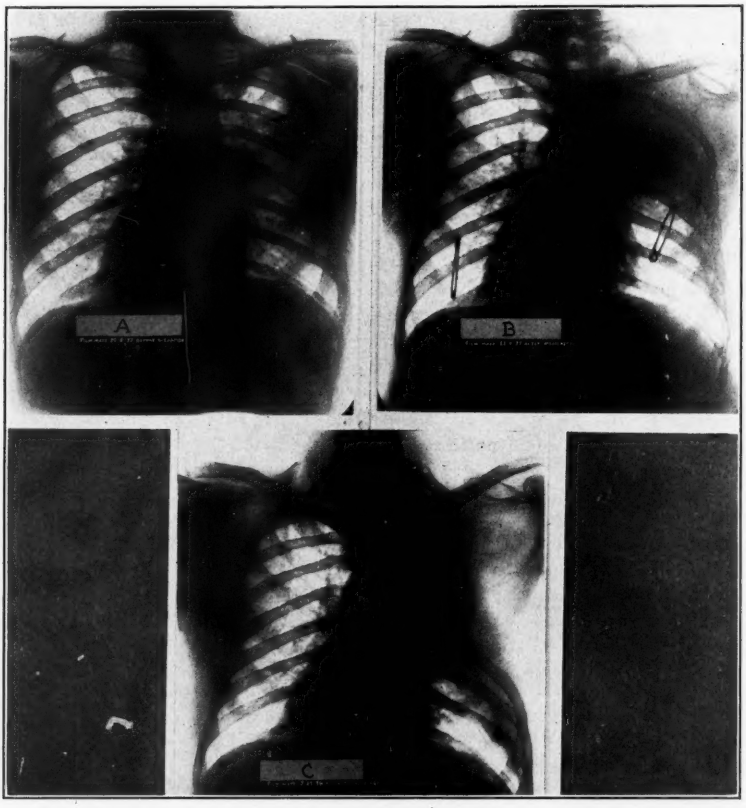

Fig. 2.—Case 10. *A*, Preoperative film Oct. 6, 1937. *B*, Film made twenty-three days after Stage 1, the apicolytic and myoplastic stage. Note complete resection of ribs two and three through the anterolateral incision. Note also the questionable spread on the left side, which caused postponement of Stage 2. *C*, Film made forty-five days after Stage 2, posterolateral resection of ribs four, five, and six. Rib one remains intact. Marked improvement on the left. Sputum negative on concentration for more than two months.

months. All wounds well healed. Opposite lung: very slight increase in disease after Stage 1 (Fig. 2 *C*); now regressive. Slight scoliosis. Arm function: lateral abduction 85° (Fig. 3). Vital capacity: before, 2550 c.c., after, 2500 c.c.; 2.0 per cent loss. Patient still in hospital under medical supervision.

Result: Questionable.

RESULTS

In summarizing the results of this operation, the benefits have been minimized rather than exaggerated, as it is too early to arrive at final figures: apparently successful, 3; questionable, 1; unsuccessful, 4; died, 2; total, 10.

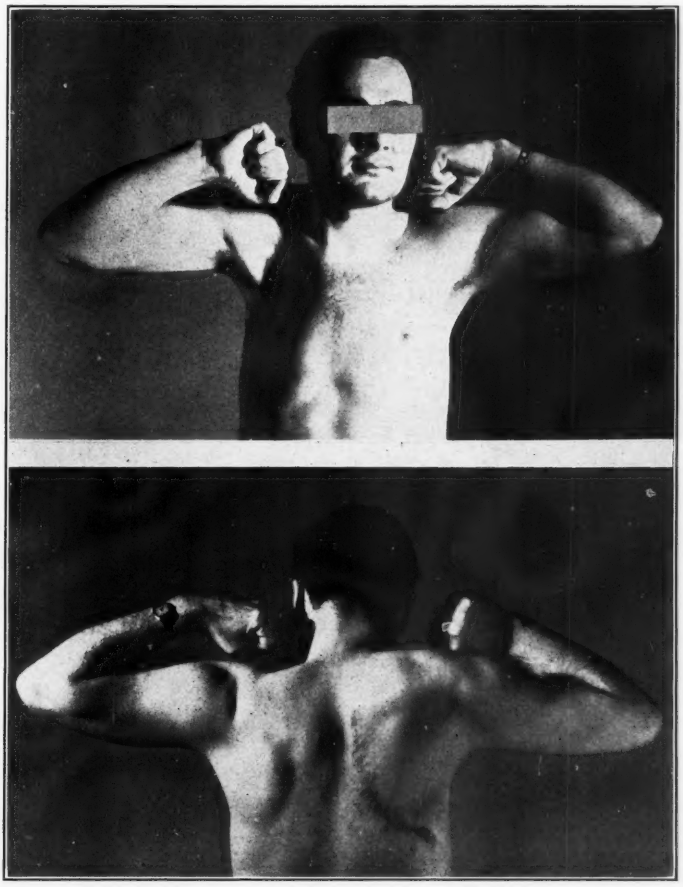

Fig. 3.—Case 10. Anterolateral scar and appearance of right chest. Posterior scar and arm function.

No case was classed as "apparently successful" until all the following requirements had been met: x-ray evidence of cavity closure, sputum negative on concentration for three months, wound completely healed, patient discharged from surgical care and returned to routine medical supervision. Three patients (Cases 6, 7, and 9) have satisfied all those conditions, and in two instances the sputum has not only

been negative on concentration but negative on culture as well. (In the New York State District Tuberculosis Hospitals cultural sputum studies, under the supervision of Dr. Max Pinner, are considered superior to animal inoculations.)

One patient (Case 10) has been classed "questionable." In this instance all the requirements for success have been satisfied, but there is a possible minimal spread to the opposite lung (Fig. 2). It is within reason to expect that he will eventually be included in the successful group.

Four cases have been classed "unsuccessful" for one or more reasons. In two (Cases 1 and 4) the sputum has been negative on concentration for five months or more, and there is no evidence of cavity by x-ray or physical examination, but there remain sinuses in the scar of the myoplastic stage (in both cases, Stage 2). Tubercle bacilli have been recovered from both of these sinuses. In the other two cases the wounds are well healed, but the sputum remains positive on direct smear. One of these patients, whose disease was of twelve years' duration (Case 3), has a residual uncollapsed cavity, much smaller than it was originally. The other patient (Case 2) has no demonstrable cavity by x-ray film, but suffered reactivation of contralateral disease, and may also have tracheobronchial tuberculosis.

One death (Case 5) could be properly attributed to the operation. This patient had tuberculous empyema with mixed infection and bronchopleural fistula, as well as extensive honeycombing of the adherent apex on the empyema side. The apicolytic and myoplastic phase of the operation was carried out at the second stage. Great technical difficulties were encountered, wound infection occurred, and from that time the patient's course was progressively downward until his death four months after the myoplastic stage. The other death (Case 8) was due to spontaneous pneumothorax on the contralateral side thirteen days after the myoplastic stage, progress being in all other respects satisfactory. This death should not be attributed to the operation per se.

Postoperative complications of minor consequence include: retention of bronchial secretions (Case 7), minimal spread to the opposite lung (Case 10), shock (Case 7), and wound infection (Cases 2, 6, and 9). Complications serious enough to defeat the object of the operation but not cause the death of the patient include the reactivation of disease in the opposite lung (Case 1) and tuberculosis of the chest wall (Cases 1 and 4). Complications contributing directly to death of patient include wound infection (Case 5), acute spread to the opposite lung (Case 5), and spontaneous pneumothorax on the opposite side (Case 7).

DISCUSSION OF RESULTS

The results, as tabulated, are not brilliant; on the other hand, all the patients presented difficult problems. Would the results have been better if a conventional thoracoplasty had been performed?

Of the eight survivors, six obtained cavity closure and five obtained sputum conversion. One failure to obtain sputum conversion is chargeable directly to an uncollapsed cavity, the others are due in whole or in part to disease in the opposite lung. In three instances contralateral disease was reactivated, once to an apparently negligible degree and without serious import to the patient and twice to a degree that has defeated the object of the operation.

Two tuberculous sinuses and one of the two deaths are chargeable to the operation. In all three of these patients the apicolytic dissection must bear the brunt of the blame. If there had been no such dissection there is little likelihood that either of the sinuses would have formed, or that the infection which eventually led to death would have occurred. Another factor may be in part responsible for the sinuses. The retention of the first rib may have created a space which could not readily be obliterated by any available soft tissue.

The retention of the first rib, on the other hand, has probably lessened the incidence of scoliosis. Of the eight survivors, four have no scoliosis and two have but slight scoliosis. In comparison, patients subjected to conventional thoracoplasties in the same hospitals have shown a higher incidence of scoliosis. If the retention of the first rib proves to be a causative factor in the production of tuberculous sinuses, there will be compelling reason for its resection. If, however, there is no reason to believe that it is a factor in sinus production, the prevention of scoliosis plus the technical advantages already mentioned argue in favor of leaving the first rib undisturbed.

Arm function was never seriously hampered; there was always a progressive improvement with time. The average loss in vital capacity, in five patients, amounted to 13.3 per cent. One patient has already returned to economic independence; the other survivors are still under medical supervision.

It is significant to note that all the unsuccessful results and the death attributable to the operation occurred in the first five cases; the second five cases include all the successful results, the questionable result, and the death attributable to spontaneous pneumothorax. The ultimate results improved as familiarity with the many technical details increased.

SUMMARY

The subject of "apicolytic thoracoplasty" has provoked considerable discussion. The reader is again referred to Alexander[1] for the arguments pro and con. It is true that the simpler the operation, the less will be the mortality and morbidity risk; likewise the less will be the probability of success. The more elaborate the operation, the greater will be the associated risks; also the greater will be the likelihood of success in difficult cases. If a satisfactory result may be anticipated from the simpler of two operations, it would be foolish to incur the risk attendant upon the more elaborate alternative. When, however,

the simpler procedure does not offer a reasonable prospect of success it becomes proper to consider alternative measures.

In the belief that the ultimate result of any surgical intervention is determined by the success or failure of the initial operation rather than by later efforts to correct shortcomings, myoplastic thoracoplasty is presented as a useful variety of apicolytic thoracoplasty. It should be reserved for patients who can tolerate a relatively severe operation, and should only be used when such an attack is deemed essential to cavity closure.

We are indebted to Dr. Robert E. Plunkett, General Superintendent of New York State District Tuberculosis Hospitals, and to the three superintendents of the hospitals in which these operations have been performed for their helpful cooperation.

REFERENCES

1. Alexander, John: The Collapse Therapy of Pulmonary Tuberculosis, p. 378, Springfield, Ill., 1937, Charles C. Thomas, Publisher.
2. Alexander, John: Ibid., Chapter XXIV.
3. Alexander, John: Supraperiosteal and Subcostal Pneumonolysis With Filling of Pectoral Muscles, Arch. Surg. **28**: 538, 1934.
4. Archibald, E. W.: Extrapleural Thoracoplasty and a Modification of the Operation of Apicolysis, Utilizing Muscle Flaps for Compression, Am. Rev. Tuberc. **4**: 828, 1921.
5. Ecot, F. C., and Jullien, W.: La Thoracoplastie par Voie Axillarie, Paris, 1937, Gaston Doin & Cie.
6. Semb, C.: Thoracoplasty With Extrafascial Apicolysis, Acta. chir. Scandinav. **76**: 1, 1935.
7. Shivers, M. O.: Surgical Treatment of Pulmonary Tuberculosis, Colorado Med. **16**: 27, 1919.
8. Tuffier, Theodore: Etat actual de la Chirurgie Intrathoracic, Paris, 1914, Masson & Cie.

DISCUSSION ON "EXTRAPLEURAL THORACOPLASTY IN PRESENCE OF CONTRALATERAL PNEUMOTHORAX," BY O'BRIEN, TUTTLE, DAY, AND O'CONNOR; "GIANT TUBERCULOUS CAVITIES OF THE LUNG," BY CORYLLOS AND ORNSTEIN; "EXTRAPLEURAL THORACOPLASTY," BY HEAD; "THORACOPLASTY FOR TUBERCULOSIS AND CHRONIC EMPYEMA THROUGH SHORT INCISIONS," BY WANGENSTEEN; "INDICATIONS, HAZARDS, AND RESULTS OF APICOLYSIS THORACOPLASTY," BY GALE AND OATWAY, JR.; AND "MYOPLASTIC THORACOPLASTY," BY BUTLER, SKINNER, DOUGLASS, AND MERKEL.

DR. EDWARD S. WELLES, Saranac Lake, N. Y.—I want to ask some questions and make some remarks about several of the papers, but I think it will be easier if I work backward, starting with Dr. Butler's paper, which is most recently in all our minds.

One question I would like to ask Dr. Butler is about the incision which he makes in the anterior chest wall, which would seem to me to produce lymphatic block in relation to the breast in women. When I first started to do anterior thoracoplasties ten or twelve years ago I used an incision like that to remove the front ends of the ribs and found that in women, especially those with fairly large breasts, the operation, although easily done and successful so far as rib removal was concerned, was followed by engorgement of the breast which lasted for weeks or even indefinitely, and I decided that I had to change my incision to run in a direction which would not cross the lymphatic channels running from the breast to the axilla.

The same question arose in looking at Dr. Head's incisions which, on his lantern slide, were marked *B* and *C*, I believe, which also went across in the same direction as Dr. Butler's.

As Dr. Butler mentioned, years ago Dr. Archibald used a muscle flap in doing an apicolysis without rib removal; that is, one small portion of rib was removed and the apicolysis done, and the plombierung was done with a piece of the pectoral muscle. The trouble with that operation and the reason it was given up was that the muscle invariably atrophied and reduced in size so that it failed to fill the cavity which had been produced by the apicolysis. I think Dr. Butler really has something to add to our system of patterns now, because enough rib is removed to reduce that space in most cases completely, whether the muscle is put in there or not, but if we can add the muscle as a stabilizing influence all the objection to the Archibald operation is removed, and Dr. Butler's procedure sounds like a valuable one. As he says, his number of cases is too small yet and his results are not extremely impressive, but it sounds like a very good pattern.

I think Dr. Gale's paper should be taken very much to heart by everyone here because it brought out the dangers and the failures and the bad results of apicolysis. There is always a great tendency, as soon as a new measure or a new pattern is brought to the attention of the group, to plunge right in and use it routinely, and in practically every case it seemed to me a year or more ago that in spite of my first enthusiasm for Karl Semb's operation we were going to have to alter our opinion about it as time went on and as we gathered some experience with it. I think a good many of us have altered our opinion and that the operation is not one to be used routinely; cases must be chosen carefully, and the operation should be employed or not employed according to each individual case and according to the progress that case makes while he is being operated upon. I think there are some cases in which the operation may be chosen at the start, but after the operation has proceeded the condition of the patient may not warrant the further trauma which the operation entails, and a note of caution should be sounded here please to take seriously Dr. Gales's results and think before you leap into apicolysis.

I wish that more men who show x-ray results of thoracoplasty operations would show better x-rays. It is the easiest thing in the world. This is not a criticism, mind you, of any person who has shown x-rays today, but everybody that has shown x-rays in the last ten years. It is the easiest thing in the world to show a case before operation with a large cavity present, then show the case after operation with no cavity showing. You can do that with any case you do, whether there is a cavity remaining or not, by a simple process of poor x-ray technique. You rarely see a case shown on the screen at these meetings in which really good x-ray technique is apparent with the use of the Bucky diaphragm or deliberate overexposure of the operated side with normal exposure of the unoperated side. X-rays of that type so commonly bring out residual cavities lying in back of the transverse processes or, if those are removed, back of the spine itself, that I would like just to enter a plea that more care be taken in showing x-rays of that kind.

Possibly the men themselves at home are making those x-rays. I can't believe that they are not, but it isn't apparent to us when we see them on the screen that that technique is being followed. When I look at one on the screen and it is described as complete closure of the cavity I can't help having in the back of my mind an element of doubt as to whether the cavity is really closed or not. Of course, negative sputum is a better answer.

Dr. Coryllos' paper was a very stimulating and interesting one, but I am not sure whether Dr. Coryllos has really accomplished something or not. He certainly gives us something to think about. As he said himself, it is a clue to something to do. I have done a few operations of muscle transplantation right into the opening of a cavity, opening it widely and putting muscle in, as Dr. Coryllos has done, not with quite the same technique but the same general principle. None of mine

worked. One patient almost died, and although still alive, she still has a bronchial fistula. The others died. I haven't felt that the operation was worth doing yet. Still, it certainly is worth thinking about.

I can't understand Dr. Coryllos' theory that if a bronchus is closed and air is excluded from the cavity, the tubercle bacilli will die because they are aerobic organisms. I am not sure Dr. Coryllos said just that, but that is the impression I got. If they die from the lack of free oxygen in air, how do they live in closed spaces where no air is available? Undoubtedly it is from oxygen brought to them in the blood stream. There must be oxygen supplied them in the blood stream of the cavity wall. Maybe bacilli will die in free secretion in the cavity, but why won't they go on living in the cavity wall? That theory confused me.

DR. HERBERT WILLY MEYER, New York, N. Y.—In the early history of extrapleural thoracoplasty at the Lenox Hill Hospital in New York City, the late Dr. Willy Meyer, in 1911, tried to get permission to admit tuberculous patients to the hospital. The patients were sent from outside sanatoria, but on account of the ruling of the State Board of Charities admission was not permissible, and the Board of Trustees, therefore, did not allow tuberculous cases to be admitted to our general hospital. Therefore, from 1911 on he was unable to operate for tuberculosis, but he gained his experience in operating for extrapleural thoracoplasty in advanced bronchiectasis, and operated upon seven patients from 1909 to 1918.

Finally, in 1918, the Board of Trustees and the Superintendent of the hospital gave him a room on the top floor of the hospital, in which he was permitted, in a secluded corner, to admit a patient with active tubercule bacilli. This patient, sent by Dr. Baldwin from Saranac Lake, had active tuberculosis, positive sputum, and ran temperatures up to 102° and 103° F. They had attempted pneumothorax in Saranac Lake, which was unsuccessful, and as a last resort he was sent for extrapleural thoracoplasty.

He was operated upon on Nov. 18, 1918. In the first stage, under local anesthesia, as advised at that time, the lower ribs were resected first. He made an uneventful recovery, and seventeen days later, again under local anesthesia, the upper ribs, up to the second rib, were removed.

An x-ray picture of this patient taken in 1917 shows multiple cavities scattered throughout the entire left lung.

In the first stage operation the lower ribs were resected first, and then the upper ribs were resected. Fairly large sections, for that period in the history of the operation, of the second, third, fourth, fifth, sixth, seventh, eighth, ninth, and tenth ribs.

Some of the older members of the American Association for Thoracic Surgery will recall that the patient was presented at the second annual meeting at Atlantic City. He wrote in a letter from Saranac Lake that as he walked along the streets of Saranac Lake he was pointed out as "The Ribless Wonder"; we must remember that that was in 1919, which is only a short time ago. He is still alive and well. He lives in El Paso, Texas, is the cashier of a bank, fully rehabilitated, married, and has a family. He went to the Mayo Clinic lately on account of migraine headaches, and Dr. Harrington, our president, was kind enough to send me an x-ray picture, at my request, which they took of this patient at the Mayo Clinic a few months ago, to show what the late result (20 years) of this thoracoplasty has been.

The x-ray picture shows a very good collapse on the operated side. It is interesting to note the amount of deformity of the spinal column. We do not know the exact cause of that because, in this instance, rib resection was not as extensive as we do now; there was no resection of the transverse processes. Still you see the amount of deformity of the spinal column but with a good collapse, with negative sputum and no cough—an arrested case.

As we pass on to the present, we perform these operations in our general hospital more frequently than was the case in those earlier years. We had an unusually high percentage of empyema cases. We had seventy-three patients in all who were operated upon from March 1, 1918 to March 1, 1933. Twenty per cent of all of these patients were operated on primarily because they had tuberculous empyema. Of these fifteen cases, seven had arrested pulmonary disease, four of them had quiescent pulmonary disease, and four had active pulmonary disease. Five of them had complicating bronchopulmonary fistulas.

In the results, apparently two are cured, nine have open empyemas, and four are dead.

The one point we would like to bring out is that perhaps it would be wiser, when we see after artificial pneumothorax that the lung which has the cavity in it does not collapse but that the good part of the lung does collapse, not to continue with pneumothorax too long, but to send these patients earlier for operation.

In the entire series we have had seventeen deaths, 23.3 per cent. In the early period, before our chest service was organized, we had twelve cases with eight deaths, or 66 per cent. Those were the patients sent to the hospital from out of town sanatoria, all of them bad, all of them sent as a last resort where nothing else could possibly help. Since Dr. Thorburn and Dr. Riggins have organized our chest service, we have had sixty-one patients (1933 to 1938) with nine deaths, or 14.7 per cent. Thus, you see the improved result after conferences between surgeons and medical men in which the cases were more carefully selected for thoracoplasty.

We had 135 operations on these seventy-three patients, with ten operative deaths, 7.4 per cent. In the early series there were seventeen operations with four deaths, or 23.5 per cent. Since the chest service was organized, there have been 118 operations with six deaths, or 5 per cent.

In the last two years, there were thirty-five operations in 1937, and eleven so far this year, with no operative deaths.

If we analyze the deaths in the early series we will find that many of them died in the early period after operation. One of them lived for eight years and that patient, a hopeless case, probably nowadays would not be operated upon. However, with operation that patient lived eight years and then died of a reactivation of a quiescent tuberculosis.

Since the chest service was organized we have had nine deaths: tuberculous bronchopneumonia, transfusion complication in an empyema case, cardiorespiratory failure, embolism proved by autopsy, tuberculous bronchopneumonia in two cases, spread to opposite side in two cases, and spread of tuberculosis.

Under rehabilitation, twenty-seven cases are back at work, nine are fair, five poor, two of them not known, eleven still at the hospital, two are worse since operation, and seventeen are dead.

I would like to bring out one more point, and that is the point Dr. Welles brought out, viz., the poor x-ray pictures that we have seen. As we now think of the past of this operation, as we think of the present, and as we think of the future, with the various pattern operations that have been mentioned and have been shown to us this morning, it will probably be wise if we will try to select the operation according to the individual case, and in that it will be of great importance to try and get a better preoperative diagnosis so that we can choose our operation for the type of case that we have to deal with.

In the past year, 1937, they have developed a new type of x-ray apparatus in Germany for the study of the chest and other parts of the body, but we are especially interested in chest. It is interesting to note that this type of x-ray apparatus was also discovered independently here in our country by Mr. Kieffer in Norwich, Conn. He took out a patent on this, which was granted in 1934, but

the Germans have developed this on a commercial basis and have put it on the market, so that it can be made use of practically at this time.

Here you see an apparatus which is called the Tomograph, in which the tube of the x-ray, during the exposure, moves in a semicircle, and while the tube moves, the plate under the patient moves in the opposite direction. That makes it possible to take plane x-ray pictures of a thickness of about 2 to 3 cm. at different distances from the anterior or posterior part of the chest. In this picture you see the patient lying on the table.

This is put out by the Sanitas firm.

In the Planigraph, which is the same principle, the patient stands erect, and some of the German tuberculosis specialists prefer this apparatus because they can fluoroscope the patient with the patient standing, as they are accustomed to do. This tube moves up on this slide while the plate moves down.

Here is a picture, for instance, of the sternum, with the clavicle. Here you see a chest with cavity in which the clavicle completely disappears, showing only the cavity. Here you see the cavity at 12 cm. depth. Here you see it at 14 cm. depth, with a cavity lower down, not seen on the plate taken at 12 cm. And here you see it at 16 cm. depth, a cavity showing on the opposite side of the chest, so that one can definitely determine at what depth in the lung these cavities are situated, and one can therefore definitely determine how best the specific case should be operated upon.

DR. EVARTS A. GRAHAM, St. Louis, Mo.—Just with reference to the last remarks of Dr. Meyer, I would merely like to say that Mr. Kieffer, to whom he referred as having obtained the first patent on this general principle, in 1934, went beyond the principle which he illustrated here as being put out by certain German manufacturers and devised a machine known as the laminograph, which was made and set up in the Mallinckrodt Institute of Radiology at St. Louis under the direction of Dr. Sherwood Moore, where it has been for about a year and a half. I understand that one of the x-ray commercial houses is going to manufacture and sell the apparatus at a comparatively low cost. The laminograph is superior to the German tomograph because, by utilizing all planes of the body, it will do everything that the tomograph will do and a good many things besides.

DR. P. W. GEBAUER, Cleveland, Ohio.—With reference to Dr. Tuttle's paper and bilateral collapse in general, I should like to say that last year at the meeting of this Association at Saranac Lake I was deeply impressed with the contribution of the late Professor Jacobaeus. In the last year I designed and, with the aid of the American Anode Company, constructed a flexible rubber catheter which could be used for this same determination. I was a little surprised that no one had mentioned it at this meeting today in regard to collapse treatment.

In 1936 and 1937 thirty per cent of our thoracoplasties had contralateral pneumothorax. Perhaps a good deal more would be operated upon if we were not hesitant because of a rather low vital capacity before operation. I have used this catheter; the patient can sit up or lie down during the determination, and the McKesson Appliance Company, of Toledo, Ohio, has loaned me two spirometers. So just as Professor Jacobaeus did, the function and capacity of each lung can be determined separately and simultaneously. We have found that the x-ray pictures are often misleading, as Dr. Jacobaeus found. I have a sample of this catheter with me and I will be pleased to demonstrate it to anyone who would be interested in seeing it.

DR. ROBERT GLEN URQUHART, Norwich, Conn.—I listened with a great deal of interest to the two men who previously discussed this paper regarding the Laminograph. The Laminograph was developed at our institution, and Kieffer works with us there.

I would also like to say, in regard to Dr. Graham's remarks, that Kieffer has left now with this machine, which not only takes different planes of the body but also may be used as a therapy machine on which they can concentrate the rays on different parts of the body, too.

DR. CAMERON HAIGHT, Ann Arbor, Mich.—Dr. Butler asked if I would mention the pectoral muscle transplant operation which Dr. Alexander described four years ago as having been useful in rare instances in the preceding years. Since the publication of the article, however, he has found no occasion to use it. The operation is a subcostal supraperiosteal implantation of the pectoral muscles beneath the ribs anteriorly. It necessarily requires that the patient for whom this operation is used must have an anterior cavity. We find that cavities are situated solely at the second and third ribs in very few instances. Furthermore, the collapse with the pectoral muscle operation is much less than with thoracoplasty. Another disadvantage of the pectoral muscle transplant in this region is that the muscle atrophies and the collapse eventually is not as great as it was immediately after operation.

Dr. Welles, in his discussion, spoke of edema of the breast following anterior thoracoplasty. In over 100 anterior thoracoplasties in conjunction with the posterior operation, and using an anterior axillary incision which extends only slightly above the axillary border of the pectoralis major, we have not seen resulting edema of the breast or arm. In one or two instances transitory edema of the breast has occurred following the application of an anterior compression brace, but the edema has promptly subsided following removal of the brace. By use of the lateral preoperative roentgenogram of the chest, as advocated by Semb, it is possible to localize cavities better than was the case several years ago, and with the improved preoperative localization of cavities, we have been able to obtain a better selective collapse. As a result we have found that the number of supplementary anterior thoracoplasties have been fewer than previously.

I was greatly interested in Dr. Coryllos' presentation and I would like to refer to an old operation which has been virtually abandoned, i.e., cavity drainage. We have seen several instances of large tension cavities, two of which had secondary infection within the cavity. One of these patients had an uneventful convalescence following a first stage thoracoplasty of two ribs. On the twentieth postoperative day the temperature suddenly rose to 103.4° F. The cavity was drained on the following day, and foul pus-containing secondary organisms were found. A spread in the contralateral lung developed, and the patient died one month later. In another patient, secondary infection and retained lipiodol, which had been injected transpleurally, were present in a tension cavity. As the patient was toxic as a result of inadequate intrabronchial drainage, thoracoplasty was judged to be too hazardous. Accordingly, the cavity was drained externally and the patient's condition promptly improved. After a wait of three and one-half months, thoracoplasty was begun, and the convalescence following operation has been highly satisfactory. In a third and earlier case, in which the contents of the cavity unfortunately were not cultured at the time of the drainage operation, a tension cavity which involved almost the entire left lung was present. It was reduced only partially in size by a seven-rib thoracoplasty, at which time the patient developed a fever of 103° F. as a result of retention of secretions within the cavity. Drainage of the cavity was then undertaken; prior to drainage the residual cavity measured 6.5 cm. in breadth and at least 10.5 cm. in length. Immediately following external drainage the cavity became rapidly smaller, and the toxic symptoms promptly subsided. A roentgenogram nine days after operation showed the cavity to be less than half the predrainage size. Complete closure of the cavity and negative sputum have occurred following further costal resection. In these cases, cavity drainage has been used not as a procedure of election but as a procedure of necessity because of the toxic symptoms that were present.

DR. EDWARD S. WELLES, Saranac Lake, N. Y.—May I have just one-half minute to correct a false impression I gave Dr. Haight. I did not speak of edema of the arm but of edema of the breast, produced by an incision in the direction of Dr. Head's and Dr. Butler's. For a long time I have used the incision that Dr. Haight mentioned, of an anterior axillary line running more or less parallel with the back of the pectoral muscle. That is almost exactly at right angles to Dr. Butler's incision, which runs from the clavicle down to the axilla. That is the one I think will produce edema of the breast. The other incision doesn't produce edema of the arm or breast or anything. It is perfectly all right.

DR. J. J. SINGER, Los Angeles, Calif.—I am very much interested in Dr. Coryllos' presentation. Those who have watched x-ray evidence of cavities have been very frequently misled as to whether a cavity was larger or smaller, or whether it disappeared. We were rather surprised, many years ago, to find a young man who had had a rather large cavity, and after a period of one month of rest his cavity disappeared. His sputum disappeared, and he became clinically well. About six months later someone told him that if he would take sun baths he would be a very well man, so he lay out in the sun for about eight hours. He developed a hemorrhage that night, came into the clinic, and we found the cavity in the same place, the same size as it was previously.

That opened up an avenue of thought as to whether that cavity had ever disappeared. It was difficult to arrive at an explanation at that time, but we felt that possibly the infiltration around the cavity disappeared, but not entirely, and due to excessive exposure to the sun's rays an acute process developed in the tuberculous area.

Dr. Cabot, Dr. Graham, and I, three years ago, and Dr. Neuhof, before us, demonstrated by the use of iodized oil that bronchiectasis is present in so many instances of so-called perfect collapse cases.

DR. W. H. OATWAY, JR., Madison, Wis.—I want to comment briefly on the paper of Dr. Gale, with which I was associated. One hundred cases is a small number from which to draw final conclusions, but we wish to stress that it has given us a perspective due to the almost routine use of apicolysis. It has given the opportunity to derive indications, but especially to emphasize the contraindications, and to explain rather than justify the hazards and complications.

Probably an outstanding complication has been atelectasis. The high incidence is partly due to the type of the lesion, as Dr. Gale mentioned, as well as other factors which can be realized, such as the condition of the diaphragm, the amount of bronchial secretion, anesthesia, etc., and partly due to recognition of atelectasis.

It is not a part of my privilege as a guest to stress recognition, but we have found, in two groups of our own cases that were followed along clinically in different manners, that the condition may be almost clinically silent, that it may be transient, and it may depend upon available methods for recognition. However, it is important to diagnose it and diagnose it quickly, because it can be treated well and, if it continues, it may have a bearing upon the mortality rate.

I would remind you again of the retrospective analysis of the cases, where at the present time we would do many of the cases with small apicolysis and some of the cases without apicolysis. The decreased extent of operation is important, since it may prevent an overbalancing of the value of repair and the shortening of the apico-hilar radius by the complications and reaction to a large dissection.

DR. W. E. ADAMS, Chicago, Ill.—I wish to make just a few remarks regarding the paper of Dr. Coryllos. A few years ago Dr. Vorwald, and more recently Dr. Escudero and I, worked with a human tubercle bacillus (H-37) in producing tuberculous lesions in dogs' lungs. The point I want to bring out is with regard to the ability of the tubercule bacillus to grow in an atelectatic lung. In some

of these experiments the lung was made atelectatic by bronchial stenosis prior to the introduction of the organisms. These were introduced through the blood stream and lodged in the atelectatic as well as the inflated lung, and the marked contrast between the inflated and the atelectatic was most striking. There were a few microscopic tubercle formations in the atelectatic lung. One had to search over more than one microscopic section in order to find the tubercles, whereas the inflated lung was studded with large tubercles.

In other experiments tuberculous lesions of a cavernous nature were made by introduction of the organisms through the bronchial passages. These cavities were then treated by stenosing the bronchi and producing massive atelectasis. They routinely improved, and, if left long enough, went on to complete healing. But this required many months, and as long as a year after this atelectasis had been produced, viable, well-stained tubercle bacilli were found in those lungs.

DR. WARRINER WOODRUFF, Saranac Lake, N. Y.—There were one or two questions I wanted to ask, particularly about apicolysis. From the first reports published I got the impression that it added very little to the risks of thoracoplasty but did add greatly to its efficiency, and it looked as though it were going to be a solution to many of our more difficult problems.

For that reason I was particularly delighted with Dr. Gale's and Dr. Oatway's very frank discussions of their findings because I believe their statistics coincide quite closely with my own. In other words, the operation has permitted the closure of cavities which under the older method I do not believe would have been closed. Nevertheless, there has been an increased morbidity and mortality. Whether this will decrease as I become more proficient in its use, I do not know.

I, too, have been struck by the increased incidence of atelectasis and its serious nature in certain instances. Also, I have had the impression that atelectasis was more common where the diaphragm is motionless. There is one complication which I have not heard mentioned this morning and I am wondering if any others have encountered it, namely, tuberculous infection of the thoracoplasty wound due to cutting across tuberculous lymph channels along the mediastinum. I have had one patient whose death I can attribute to that. Tubercle bacilli were recovered from the wound within a few weeks.

There is one question regarding the anterior approach as discussed by Dr. Head, Dr. Wangensteen, and Dr. Butler. I am still wondering if the anterior axillary approach as described by Dr. Welles several years ago and later modified by Dr. Alexander does not give much better exposure than the shorter incisions described this morning. With the axillary approach, few muscle fibers are cut, the pectorals being lifted forward. I am sure you can get adequate exposure to the sternum for all except the first rib and I have even removed the anterior end of this rib from this incision but do not like to.

I should like to ask a question of Dr. Singer. He mentioned the use of iodized oil. Has he seen any instance in which its use has been followed by a spread of the tuberculosis, particularly in an individual where the original lesion was not thought to be acute?

DR. R. H. OVERHOLT, Brookline, Mass.—There have been so many interesting points mentioned that it is difficult to cover them in a discussion. I believe I should say I am a bit disappointed in some of the results that have been reported in employing apicolysis with thoracoplasty.

A year ago we analyzed results in over 250 cases. A report was published in the February issue of *The American Review of Tuberculosis*, and I think it is fair to say our results during the past year have not been dissimilar, so that we still are inclined to continue to use mobilization as a routine part of thoracoplasty.

I feel that the introduction of these newer methods using smaller skin incisions and smaller muscle incisions may possibly be a step back rather than a step forward.

Whenever possible, it is preferable to have an adequate exposure in doing any surgical procedure. We know that the shock of thoracoplasty is not so much in the amount of skin divided or the amount of muscle divided, but in the amount of rib resected at one time, which to my mind does not create a problem of improved methods of stripping a long segment of rib but the problem of holding back and making the decostalization less extensive than it was in the past.

Unfortunately in Massachusetts about 85 per cent of all patients admitted to public sanatoria have far-advanced disease, and a great proportion have bilateral disease. It is necessary, if we are going to give them permanent collapse, to make this collapse as selective and conservative as possible, so we have come to use a selective type of thoracoplasty with lung mobilization either on one side or on both sides, carrying out a limited upper stage thoracoplasty of two and one-half ribs, always leaving the anterior portion of the third rib and then delaying further operation at least six weeks. Then the patient has recovered from the effects of the first operation. The blood lost at the first operation has been replaced, the patient has been out of bed, has been walking about, and then is in a fit condition to undergo a second operation if a second operation is necessary. Then it is up to the surgeon to plan his operation at the time of the second stage so that he produces a satisfactory collapse as a final result.

I believe that by the use of saline in the apicolytic pocket at the time of the first operation, and also the excision of scar in the region of the bridge formed between the area of the first operation and the second, it is possible to have just as satisfactory a final collapse as we provide by the rapid interval of stages. We rarely lose a patient after the first stage. Most of the deaths in our early series were after a second or third stage, and these occurred when the various stages were carried out in rapid succession. So that I think we should focus our attention not upon the skin and muscles or ribs, possibly, but more upon the type of selective collapse of the diseased lung and the conservation of healthy portions of lung.

I cannot give exact figures, but in the paper that was published I think the average number of ribs resected in this large series of cases was a little under six, including all thoracoplasty done, and the average number of stages per operation was 2.2 per cent, and the mortality was 2 per cent for the unilateral cases and 8 per cent, I believe, for the extensive bilateral cases or progressive lesions, patients with exudative disease, with cavitation, with fever, etc.

DR. POL N. CORYLLOS, New York, N. Y.—We certainly had today a most interesting symposium on surgical treatment of pulmonary tuberculosis.

We all agree today that thoracoplasty should be as selective as possible and we should proceed with the constant pre-occupation to conserve the function of as much healthy parenchyma as is compatible with adequate collapse of the diseased portions of the lung. As for the technique of thoracoplasty I do not think that it is, as yet, completely and definitely standardized. Useful modifications are suggested continuously and we must consider them very seriously because they may be of use in special cases. As a general rule I think that we should resect completely the first two ribs and sufficient lengths of the third and fourth, according to the size of the cavities.

Regarding the apicolysis of Semb, I believe that it increases the operative time and operative shock. It is not an indispensable procedure in cases with small cavities or simply moth-eaten apex. But it is an excellent addition to thoracoplasty in large and especially in giant cavities. I am convinced that extensive apicolysis should be carried out in these cases because, in my cases at least, it has doubled the percentage of success.

I do not use any saline solution to fill the extrapleural space after apicolysis. Very shortly after operation we find this space filled up with serohematic fluid which is gradually absorbed within three to four weeks.

I perfectly agree with Dr. Graham and Dr. Churchill that in all thoracic operations we must avoid mutilating or deforming procedures. Such procedures give a black eye to thoracic surgery and retard its development.

I was particularly thrilled with the paper of Dr. Tuttle and Dr. O'Connor. In 1936 in a paper on the management of bilateral cases (this JOURNAL) Ornstein and myself insisted upon the usefulness of contralateral pneumothorax as means of controlling the lesion while thoracoplasty was carried out on the other side. I perfectly agree with the authors that our efforts today must be directed not to the improvement of our statistics by restricting the limits of operability but to the saving of more patients by deciding on procedures which will allow us to extend the limits of operability and take in as many bilateral cases as possible. Contralateral pneumothorax and perhaps also extrapleural pneumothorax are for the time being the methods which are of great help in operating upon cases so far considered beyond any surgical help.

DR. L. R. DAVIDSON, New York, N. Y.—I should just like to leave one point of warning. If my recollection serves me rightly, I believe Dr. Butler indicated that in one of his cases where he had a contralateral pneumothorax the patient developed a spontaneous pneumothorax, and death followed. This applies to Dr. Tuttle's paper.

For some time we have been performing thoracoplasties with contralateral pneumothorax. We unfortunately permitted such a patient, the sputum being negative, apparently arrested, to go home. The patient developed a spontaneous pneumothorax and died. Today we do not permit any thoracoplastic patient, regardless of the x-ray picture, to leave the hospital if he has a contralateral pneumothorax. The reason is obvious. We do not know when that patient will develop a spontaneous pneumothorax. If that patient is in the hospital he is under competent control and can have continuous deflation.

As regards the papers of Dr. Head and Dr. Wangensteen, I can only say that one's judgment is perhaps dependent upon his early training. Having trained in part with Dr. John F. Erdman of New York City, I know only one thing about surgery: one must have adequate exposure and the ability to control any hemorrhage and any complication that arises at the time of the operation. Because the exposure is inadequate, I feel these miniature incisions have no place in the thoracoplastic operation. We cannot obtain exposure of the scalenus and serratus, and without that, as I think Dr. John Alexander, in the first article of the second volume of the JOURNAL OF THORACIC SURGERY, showed, we cannot get complete excision of the ribs.

Since we have been talking about plans, schedules, and programs, may I indicate that I have a pattern about which I know very little. In three cases, one patient died from a contralateral bronchopneumonia, one recovered with a negative sputum, and the other persisted with a positive sputum. The pattern is this: the Semb operation must be performed primarily. That is the first stage. Then as one collapses the cecum in cecum mobile, in somewhat the same fashion one inserts a purse string in the endothoracic fascia and adequately depresses or invaginates the lung apex. As yet I do not know what value this procedure possesses. I merely submit it as another pattern.

Finally, relative to the question of Dr. Woodruff, in the early days of the Semb operation we too found lymph glands at the level of the second rib in the costovertebral gutter, and on occasion, being of an inquisitive turn of mind, we removed them. We have abandoned this procedure for the same reason Dr. Woodruff has. Upon cutting across lymph vessels, tuberculous infection of the wound resulted in a few instances.

DR. J. J. SINGER, Los Angeles, Calif.—I would say the use of lipiodol, or any iodized oil, should be limited to cases where it is absolutely necessary to know

what is present in the lower lobe and the reason why the patient doesn't get well. The indiscriminate use of the oil certainly should not be allowed.

DR. WILLIAM M. TUTTLE, Detroit, Mich.—We have had just one patient who developed a spontaneous pneumothorax two or three days postoperatively. In that case, by introduction of a catheter, the lung was re-expanded, and no fatality occurred; the cavity remained closed.

We have had three other cases in which there was a spontaneous pneumothorax, but the emergency was handled without the introduction of the catheter. I think what Dr. Davidson has said about keeping these patients in the hospital until the contralateral lung has re-expanded is probably right; although if we worked on the same basis, we would keep all bilateral pneumothoraxes in the hospital until re-expansion has occurred, and that is not done.

Practically all of our thoracoplasties have phrenic nerve paralysis on the side of the thoracoplasty at the time of operation. The number of cases which require postoperative bronchoscopy is no greater than in a similar group in other clinics where perfect freedom of motion of the diaphragm on the thoracoplasty side is present. We feel that the cause of atelectasis, in the first place, is the type of disease, and secondly, the condition of the bronchus before thoracoplasty is done. Atelectasis has developed, in many patients, when it might have been avoided had the operator known the condition of the patient's bronchus before thoracoplasty was done. We routinely bronchoscope all of our thoracoplasties to determine that point alone.

The other papers I enjoyed a great deal, although I do think that operations with small incisions are definitely dangerous. Oftentimes, I experience enough trouble stopping bleeding when I have the thoracic cage well exposed, and I cannot see how bleeding can be controlled when the operator is working through a small incision and under a large flap of skin and muscle.

The instruments used by the advocates of the small multiple incision type of thoracoplasty are similar to the small periosteal elevators of Dr. E. J. O'Brien. While this type of instrument does not cause as serious injury to the intercostal vessels as do the larger periosteal elevators, still injury with bleeding does occur. Such operative procedures foster what appears to me to be a method of blind surgery, and carry with them great danger.

DR. POL N. CORYLLOS, New York, N. Y.—I want to thank the discussers for their kind remarks and their constructive criticism. Dr. Ornstein and myself wanted to present to you our ideas upon the mechanism of closure of tuberculous cavities and the importance of the relationship of the condition of the bronchus on the course of these cavities.

We state in our paper that following closure of the draining bronchus the oxygen of the air contained in the cavity is absorbed so that a marked deficiency of oxygen is created in the cavity, the result of which is the hampering of the growth of tubercle bacilli in the cavity and of their metabolic activity.

The objection was raised whether this deficiency could cause the death of tubercle bacilli and whether such a deficiency would really be produced, since oxygen could and should pass into the cavity through its wall coming from the venous air of the blood circulating around the cavity.

The answer to the first objection is that it has not been proved that even complete absence of oxygen could kill all tubercle bacilli. What we know is that when oxygen has been replaced by nitrogen in vitro, the respiratory quotient of tubercle bacilli falls very rapidly to zero, and they stop metabolizing. They may remain in this condition for a long time, and if oxygen is again added in the medium the tubercle bacilli may again resume their growth. This is consistent with what occurs in tubercle bacilli walled in fibrotic tubercles or lymph nodes. Recent

work seems to indicate that when smooth colonies have been submitted for some time to anaerobic conditions they grow, but when oxygen is given to them again they grow rather in rough than in smooth colonies, which would suggest loss of virulence.

Regarding the second objection, namely, whether sufficient deficiency of oxygen could be created and maintained in the cavity following closure of its bronchus, I shall answer without any hesitation that sufficient deficiency is created and it is maintained as long as the bronchus remains closed. I have given the proof of it in a paper on "Mechanics of the Cavities" published in 1936. The absorption of gases in the air trapped in the cavity takes place according to the rules which regulate the production of atelectasis following obstruction of a large bronchus or the absorption of air in subcutaneous emphysema, pneumoperitoneum, and even in closed pneumothorax. Moreover the presence of organic substances and pus, which take up oxygen, speeds up the absorption of oxygen. A similar phenomenon takes place in pyopneumothorax, in which, as I have shown in a paper published in this JOURNAL, oxygen drops below 1 per cent.

A third point which we wish to make clear is that the term "blocked cavity" is a bad term. Most workers associate blocked cavity with a closed bronchus when really the term implies an obstructed bronchus which may or may not be open. In other words, a blocked cavity may be an obstructive emphysematous or the ball-valve cavity. This cavity is not closed. Air gets in but has difficulty in escaping. At the same time a blocked cavity may be a completely stenosed one, allowing no air to enter or escape through the bronchus. Both types are blocked cavities and cannot be discussed as similar situations. A great deal of confusion is caused by the term blocked cavity.

The objective of our work was to attract the attention of our colleagues to the paramount importance of the draining bronchus of the cavities and stimulate their efforts to the research of means which may bring about their closure.

DR. JEROME R. HEAD, Chicago, Ill.—Dr. Elkin told a story yesterday about a southern politician who was asked how he stood on a certain political question. He replied, "I haven't made up my mind yet, but I warn you that when I do make up my mind I am going to be awful bitter about it." Dr. Overholt and Dr. Davidson and Dr. Tuttle are much more convinced that the operation which I have described is bad than I was that it is good; but since their discussion I have become bitter and am going to defend it.

I don't know whether it is good or not, but I disagree with Dr. Overholt's statement that the shock of thoracoplasty comes from rib resection and not from muscle trauma. One takes out just as much rib in the operation I have described, yet the reaction is actually much less. The important question is, Can one secure a satisfactory a collapse? I think that that is open to question.

The inability to sever the attachments of the serratus and the scalenus is a serious drawback. In regard to the control of hemorrhage, it is not a factor, because one just does not get hemorrhage. Hemorrhage arising from stripping of rib in thoracoplasty stops of itself. In twenty operations we have had no trouble from this source. The question will be decided by the end results of the operation and by no other method.

I think that a good many surgeons have been too willing to turn down procedures on theoretic grounds. Extrapleural pneumolysis with paraffin pack was rarely tried in this country for a great many years and has been abandoned after a very scant trial because of theoretic objections. Wölfler, who did the first gastroenterostomy, was strenuously criticized when he first reported the operation. A great many theoretic objections were raised. It was said the patient would starve to death, that intestinal secretions would regurgitate into the stomach, and that

one thing or another would happen. In his closing discussion Wölfler replied: "All theoretical objections which can be raised against the operation can be answered by the fact that it works."

I still believe that extrapleural pneumolysis with paraffin pack is a valuable operation and that there are situations in which it is the only feasible method to collapse the lung.

Dr. Welles has spoken of the desirability of standardizing the technique of x-ray examination after thoracoplasty. The standardization of sputum examination is equally important. The final test of the efficacy of the various collapse measures must be determined by the presence or absence of tubercle bacilli in the sputum. A just appraisal of their relative value can therefore be determined only if the method of sputum examination is standardized. A poor laboratory is a great source of encouragement to the thoracic surgeon; a good laboratory does much to discourage him. The method of sputum examination which is least subject to error and variation is the injection of stomach washings into a guinea pig. It would probably be well if all reports of the results of collapse therapy were based on this method of examination.

I share the skepticism which has been expressed concerning extrafascial apicolysis as described by Semb. In using artificial pneumothorax every one has had experience with the mobile mediastinum and has seen cases in which the cavity, instead of collapsing, runs away from the pneumothorax. Thoracoplasty is being done more and more frequently for earlier lesions and for those in which fibrosis has not stabilized the mediastinum. In such cases even though the apex of the lung is freed and allowed to retract toward the hilus, thoracoplasty is unable frequently to close the cavity, because the lung retracts ahead with collapse of the chest wall.

Two years ago at the meeting of this society I read a paper on the use of the paraffin pack as a preparation for thoracoplasty, stressing the facts that it both reduced the size of the cavity and so stabilized the mediastinum as to provide a firm barrier against a retreat of the lung. At the time of that meeting I had already abandoned this procedure in favor of the Semb operation. Recently I have readopted it for use in cases with large apical cavities and apparently mobile mediastinums.

DR. OWEN H. WANGENSTEEN, Minneapolis, Minn.—With reference to the problem of thoracoplasty, I believe we can all agree on one thing, viz., our objective is to secure cavity closure and convert the sputum so that it is tubercle bacillus-free. How that objective may be best attained is apparently far from being a matter of common agreement.

I must confess to a feeling of some amazement over the complacence and self-satisfaction expressed by some who have had at least as much experience with the posterior approach as I have. It is difficult to escape the impression that the magnitude of the operative undertaking, the cutting across of muscles, and the problem of hemostasis in the posterior operation can be accepted without some misgivings. I do not propose to become a medical evangelist as Dr. Head lent the impression of the role he might assume with reference to the advantage of multiple incisions. I am not here to win converts for an operative procedure. My only purpose in appearing before you is to announce that a rib may be removed by methods more simple than we are accustomed to employ in the orthodox operation of thoracoplasty. The procedure is less formidable for the patient and offers the surgeon far greater latitude in the extent of rib he will remove at any one operation than does the usual typical thoracoplasty.

Relative to the concomitant performance of extrafascial apicolysis, there are essentially two types of difficulties which hedge about it. The first concerns the increased intrabronchial accumulation of secretion which of necessity attends the

greater collapse secured by apicolysis. With frequent aspiration of the secretions after the method of Haight, immediately upon completion of the operation and during the early convalescence the problem of atelectasis offers no real difficulty. It is important to keep the patient's bronchus as dry as possible. The other difficulty relates to the dead space attending depression of the pulmonary dome. I have measured this space routinely in the anterior extrafascial apicolysis and find by pouring in saline solution that it holds from 125 to 350 c.c., about 250 c.c. being the more usual. By inspection of a skeleton or examination of an x-ray film of the thorax, it is evident that the problem of the residual space relates itself essentially to the matter of the sternoclavicular articulation acting as a fulcrum upon which the clavicle supports the shoulder and keeps it from falling toward the thorax. By shortening the clavicle, a procedure which I have not yet done, I dare say one could obliterate readily a great portion of the dead space attending extrafascial apicolysis. Dr. Butler has employed a myoplastic filling to reduce the size of this space. I have been inclined, in the main, to rely upon maintaining as strict an asepsis as one can achieve in an operation. Rib resection alone will not obliterate this dead space. It fills with a coagulum which in the absence of infection becomes organized and remains sterile.

DR. JOSEPH GALE, Madison, Wis.—I am certainly glad that no one gained the impression that we tried to present apicolysis as the universal panacea of collapse therapy. We do feel, however, that it is a useful adjunct to the ordinary thoracoplasty. We also feel that every case may demand a different type and degree of collapse. After all, any collapse therapy must be applied in sufficient amount, and the degree of collapse must be adequate to produce the desired closure of the cavity or arrest of the disease.

Due to the short time alloted to the paper, a detailed discussion was impossible. I should like to mention a word regarding tuberculous wound infection, which Dr. Davidson also discussed. During the past two years we have excised what we call a sentinal gland which lies at the extreme cupola of the lung. This gland is frequently enlarged to the size of a pecan. Sometimes it is even pigmented or anthracotic, and upon microscopic examination 60 per cent of these have been proved tuberculous. Naturally in removing the gland we have divided infected lymphatic channels. We have not had a tuberculous infection arise in any of the patients from whom the glands have been removed, but we have had tuberculous infections which have appeared late. These occurred in two instances, where we were applying the type of drainage advised by Dr. Semb, namely, a catheter for twenty-four to forty-eight hours. We watched these patients for four weeks, and both continued with an unexplained fever. They were irrigated with Dakin's fluid but the drainage was inadequate, and we violated one of the basic principles of surgery, namely, adequate drainage.

One of the patients had a severe wound hemorrhage which necessitated immediate operation. At operation we found the walls of the dead space above the lysed apex studded with tubercles. The internal mammary artery had been eroded, and this was the source of the hemorrhage. Even the walls of the great vessels were involved. The patient died a few hours later.

The second patient presented a similar picture, and also died. In this case an intercostal vessel had been eroded by the tuberculous process. In all the other patients, where we have not opened a tuberculous cavity or forced the lysis and cut or torn through peripleural tissue which undoubtedly contained tubercle bacilli, we have been able to control the tuberculous infections very easily.

There is one word I should like to say regarding postoperative compression. It is surprising to me that it has not been mentioned before, because we feel that it is just as important as, if not more so than, the thoracoplasty itself. I feel

that we owe a great deal to Dr. Woodruff, who almost ten years ago described a brace for postoperative compression. Innumerable braces had been described before that, but most of these encircled the chest, frequently limiting the motion of the good lung more than the diseased one. Dr. Woodruff's brace fitted over the shoulder in such a way that the good lung was allowed free movement but good compression was maintained on the diseased lung where it was most needed.

A short time ago Dr. John Steele, of Ann Arbor, later of Milwaukee, devised a spring brace which impinges on the scapula and beneath the clavicle. It is most efficient. I have seen one patient upon whom it was applied, and you could actually palpate the scapula through the anterior chest wall. In our series of apicolyses lung has risen about one-half interspace with previous fixation. When not fixed, it ultimately retreated to the apex.

Please remember our statistics in which we found 45 per cent of the negative gastric concentrates positive on guinea pig inoculation. This recalls the figures of Gullbring and Levin, of Stockholm, who had 348 cases with negative sputum. Of these, 55 per cent were positive to gastric analysis. How many would have been positive to guinea pig?

This brings up the question: What are we going to do with such patients? Are we going to consider them safe to return to society, because their cavity is closed and the sputum is negative? I hope not. We owe it to ourselves as well as to the patient to exhaust all of the more delicate methods of examination and find them negative before we can consider their disease arrested. A patient with a negative sputum but who still has a positive gastric or guinea pig test is as capable of spreading tuberculosis as a typhoid carrier is of spreading typhoid fever. It would seem to me that we have been too easily satisfied with our apparently good results.

DR. ETHAN FLAGG BUTLER, Ithaca, N. Y.—Edema of the breast has not been observed as a sequel to the lateral or axillary incisions described in "myoplastic thoracoplasty." There is apparently adequate lymphatic drainage medially toward the mediastinum to prevent lymphedema.

One of the ten cases reported had retention of bronchial secretions in the lower lobe on the operated side. None had atelectasis. Some years ago Duff Allen advised that "wet" cases be operated in the afternoon. We have followed that advice, and we have also freely employed bronchoscopic aspiration of retained secretions.

Personally, I would rather do a nonapicolytic thoracoplasty than an apicolytic operation; but, even more, would I rather make the initial thoracoplasty adequate than rely upon a subsequent revision. As long as the uncollapsed cavity remains as a problem in thoracoplasty it will be proper to meet unusual patterns of disease with unusual patterns of operation.

Erratum

In the August issue of the JOURNAL, page 678, Dr. Leo Eloesser is incorrectly quoted as saying in his rebuttal discussion: "I ligated the whole hilum, thinking thereby that I could ligate the pulmonary artery, the pulmonary vein, and the bronchial artery. This ligature was compressed to a degree to which everything was compressed except the bronchus. That is difficult to do, because it is easy to compress almost all the arteries without including the bronchus." The last sentence should have read: "That is not difficult to do," etc. Because of Dr. Eloesser's absence from the country he was unable to correct the reporter's transcript of his discussion.

In Memoriam

POL N. CORYLLOS
1880-1938

Dr. Pol N. Coryllos, after a brief illness, died on July 26, 1938. Death was most untimely and took from us a great scholar, a great surgeon, and a great physiologist.

Dr. Coryllos was born in Patras, Greece, in 1880. After his graduation from the College of Athens (B.A.), he obtained his medical degree from the Medical College of the National University of Athens in 1900. He then went to Paris, where he obtained his B.S., M.S., and Ph.D. from the Sorbonne, and a second medical degree from the Medical College of the University of Paris in 1914. During this time he served as Instructor of Anatomy (1902 to 1910), of Pathology (1909 to 1911) and Assistant in Gynecology (1910 to 1912) under Dr. Jean Louis Faure. In 1912, during the Balkan War, Dr. Coryllos returned to Greece as a volunteer. He was commissioned as Captain in the Medical Service of the Greek army and appointed director of an ambulance unit composed of seventy-five beds. This he organized as a mobile hospital, thus bringing the surgical hospital to the wounded, which was contrary to the established custom of transporting the wounded to the hospital. This unit established by Dr. Coryllos was the precursor of the medical organization later used during the World War. Thus was he able to treat surgically, and within a few hours after injury, abdominal, thoracic and skull wounds. His experiences were published in a number of articles and in book form in Paris in 1914, under the title of "Preventive Trepanation in Bullet Wounds of the Skull." For this he was awarded the gold medal from the Medical Academy of Paris.

At the end of the Balkan War he resumed his hospital position in Paris (1913). In 1914 he was a lieutenant in the French Army. He served at Dieppe, Bar le Duc, and Verdun from August, 1914, to December, 1916.

In December, 1916, he was sent by the French Government as a member of a special mission to Russia to organize the surgical service at the Russo-Rumanian front. He was given the medical direction of the division of Padureni, where he organized the mixed front hospitals, Russian and Rumanian.

In March, 1918, he was recalled to France, promoted to the rank of major and served at Château Thierry until the Armistice (1918).

Immediately following the Armistice, at the request of the Greek Government he was sent to Greece on a special mission to organize the

surgical service in the Greek army, where he remained until 1922, with the rank of surgeon general. During this time he devoted himself to the study of wounds of the chest, for which he had organized a special surgical center.

In 1919 he was appointed Professor of Surgery in the Medical College of the National University of Athens, from which he had graduated nineteen years before.

The decorations which Dr. Coryllos received from various countries he had served are a slight index of the value of his work during this period. From France he received the Croix de Guerre and the Cross of the Legion of Honor; from Greece the Cross of Savior (Officer and Commander), the Cross of Valor, the War Cross for Distinguished Services, and in 1936 the Gold Cross of the Redeemer (Commander); from Serbia the Cross of the Savior (Officer); from Rumania the Cross of the Rumanian Star (Officer), the Cross of Distinguished Services, and the Cross of Queen Marie; from Russia the Cross of St. George, the Cross of St. Vladimir (Commander), the Cross of St. Stanilious (Commander), and the Cross of St. Ann's (Commander).

In 1923, by which time Dr. Coryllos was in New York, he was appointed Professor of Clinical Surgery at the Medical College of Cornell University, which position he held until his death.

His activities in this part of the world were closely related to the investigation of the surgical treatment of thoracic diseases, with which specialty he finally became completely identified. At the time of his death Dr. Coryllos was Director of Thoracic Surgery at the Metropolitan Hospital and Sea View Hospital in New York and the U. S. Veterans Hospital at Castle Point; Associate Surgeon and Director of Thoracic Surgery at the Polyclinic Hospital; Consultant Thoracic Surgeon at the Broadstreet Hospital, Staten Island Hospital, Beth Israel Hospital, of Newark, and Vassar Brothers' Hospital at Poughkeepsie; Consultant Surgeon to St. Luke's Hospital at Newburgh, N. Y., and Director of Thoracic Surgery at the Manhattan General Hospital.

Mere recital of accomplished deeds gives but lip service to the true qualities of the man. To an indefatigable worker add the rare qualities of an inquisitive nature, an analytical mind, and the ability to construct. Thus we had the master man. Blessed with a rare sense of humor and friendliness he captivated all those with whom he came in contact. His life was devoted to the charitable hospital. The world is poorer for his loss.

G. G. Ornstein.

Book Review

TREIZE COUPES HORIZONTALES DU THORAX. By A. Hovelacque, O. Monod, and H. Evrard. Paris, 1938, Librairie Maloine. LE THORAX. ANATOMIE MEDICO-CHIRURGICALE. By A. Hovelacque, O. Monod, and H. Evrard. Paris, 1937, Librairie Maloine. 356 pages.

These two books by the same authors will be found to be very useful by all those interested in thoracic surgery. The rapid development of this branch of surgery has emphasized the need of a presentation of the special anatomy involved in this field. The authors have undertaken their work from a point of view which has been inspired by their own active interest in thoracic surgery. It is fortunate for other thoracic surgeons that this has been so. The numerous illustrations and their illuminating legends supply the sort of anatomic information for this region which most of the purely anatomic textbooks fail to give. The text has been admirably written and there is a large bibliography. Probably the book will be found generally more useful than the atlas of cross sections, but one work nicely supplements the other. The reviewer wishes to extend his personal thanks to the authors for their splendid accomplishment.

Evarts A. Graham.

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The 1939 Meeting of the American Association for Thoracic Surgery

The 1939 meeting of the American Association for Thoracic Surgery will be held in Los Angeles, Calif., June 26 to 28. Dr. Frank Stephen Dolley, 427 South Arden Boulevard, Los Angeles, is in charge of the local arrangements.